ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED IN 1892

VOLUME 66

Published Quarterly

BY THE

ANNALS PUBLISHING COMPANY

Business Office ✦ P. O. Box 1345, Central Station 1

ST. LOUIS, MO., U.S.A.

Annual Subscription in United States, $14.00 in Advance.
Canada, $14.40. Other Countries, $14.80.

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ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 66 MARCH, 1957 No. 1

I

OBSERVATIONS ON THE SURGERY OF THE NASOPHARYNX

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The nasopharynx, with its adjacent sphenoidal and posterior ethmoidal regions might well be described as the Cinderella of the nose and throat regions; it has always been and still remains the most difficult area to examine, and consequent upon this the diagnosis of many conditions, frequently serious, has often been incorrect and is in most cases delayed, sometimes for many months. To our shame we must admit that such incorrect or delayed diagnosis has not been of great moment because, even if the diagnosis is established, the result of treatment is all too often disappointing.

Clinical examination is generally thought to be too difficult for the student to master, so that it is unlikely to have been undertaken prior to examination of the patient by the specialist. Even at this stage things are little better, because of the difficulty of interpretation of the physical signs found at examination; and this is borne out by the variety of methods which may be employed. Digital examination, apart from being unpleasant, gives comparatively little information other than in very limited groups of cases: various types of direct speculae are likewise only infrequently of real value; the postnasal mirror, even if aided by local anesthesia and some form of palatal retraction is not really satisfactory in difficult cases because it will, in general, only provide an 'edge on' view and such a view can be very misleading. Radiology and pathological investigations are helpful and it was hoped that improved types of nasopharyngoscope might at last provide the solution to the problem, but in the view of most

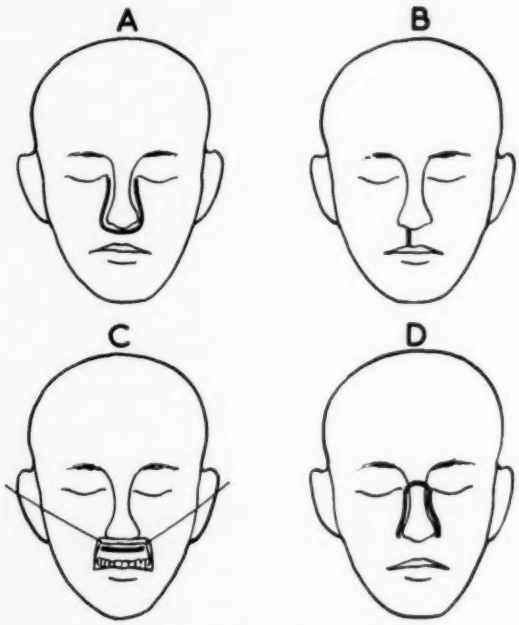

Fig. 1.—Incisions for approach to the nasopharynx. A. Dupuytren; Lawrence. B. Syme. C. Rouge. D. Ollier.

surgeons they have proved disappointing in practice and the problem of a really satisfactory method of clinical examination remains unsolved.

As a result of this difficulty of satisfactory access and exposure, treatment of nasopharyngeal conditions has lagged behind that of other regions.

The problem is not a modern one for just over 100 years ago a method of approach to the nasopharynx by lateral rhinotomy was practiced. Since that time the number of operations designed to provide access has been legion. Most have had as their primary object the removal of tumors of the maxillo-ethmoidal region, but they have also been used for nasopharyngeal fibromata or tumors of the pituitary gland and their very diversity is sufficient testimony to the unsatisfactory nature of any particular procedure advised.

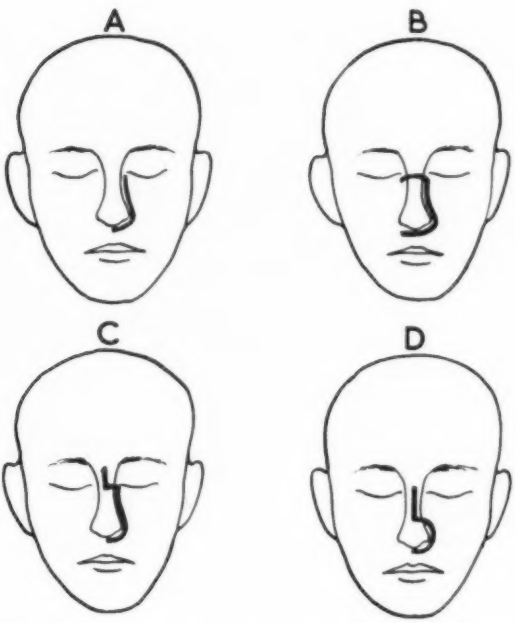

Fig. 2.—Incisions for approach to the nasopharynx. A. Moure. B. Langenbeck. C. Eiselberg. D. Chassaignac; Bruns.

These operations fall naturally into three main groups: a) those using direct access through the nasal cavity; b) transmaxillary approaches; c) approaches through the palate.

A) Access to the nasopharynx may be obtained in some cases directly through the nostril after shrinking the mucosa with cocaine and adrenalin or some similar substance; further access may be obtained by fracturing one or more of the turbinates outwards but it is not often that satisfactory exposure can be obtained by such a limited interference.

Still further exposure can be obtained by separation of the soft tissues from the margins of the anterior choana followed by removal of the nasal septum and perhaps part of the lateral walls of the nose.

Dupuytren[1] and Lawrence[2] used an incision around the nasofacial sulcus from the edge of one nasal bone to the other, detaching the

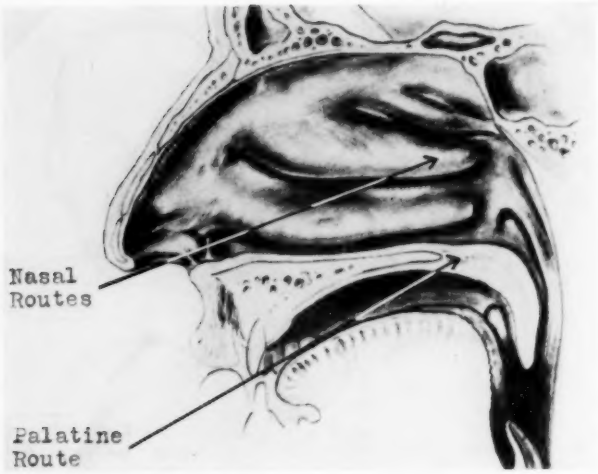

Fig. 3.—Sagittal section through the nose comparing nasal and maxillary routes with a transpalatine approach.

skin and cartilages from the bone and elevating the whole of the front of the nose (Fig. 1a).

Syme[3] divided the whole thickness of the lip on one side of the midline dissecting all soft tissues from bone on either side (Fig. 1b).

Rouge[4] and Halstead described operations employing a transverse sublabial incision to mobilize the anterior parts of the nose (Fig. 1c).

Ollier[5] used an inverted V incision turning the whole of the facial part of the nose downwards (Fig. 1d).

Moure[6] suggested an incision in the nasofacial sulcus on one side extending from the inner canthus downwards as far as the columella (Fig. 2a). Langenbeck,[9] Eiselberg, Chassignac[7] and Bruns[8] used lateral incisions of a similar nature with some modifications (Fig. 2b, c, d).

All these methods of approach give reasonable access to the anterior parts of the nasal cavities and to the sphenoidal sinus but are associated with considerable postoperative scarring and deformity

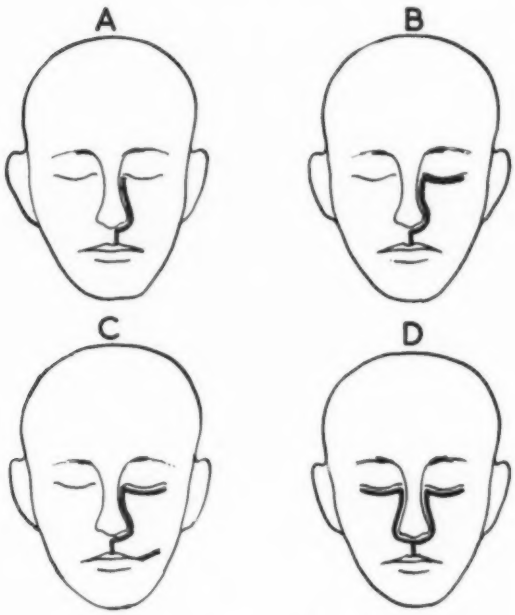

Fig. 4.—Trans-maxillary approach to the nasopharynx. A. Weber's modification of Moure's incision. B. Heath's modification of Moure's incision. C. Hugier's modification of Moure's incision. D. Cheever.

and provide a comparatively poor exposure of the nasopharynx. This is obvious if one looks at a sagittal section of the nose (Fig. 3).

B) Almost as many varieties of approach through the maxilla have also been used. These fall into two groups according to whether the incision is made sublabially through the alveolar sulcus or through the skin of the face.

Moure's incision may be used to provide an approach through the antrum as well as through the nasal cavity and modifications of this incision have been described by Weber[10] who divided the lip in the middle line so as to reflect the lower part of the cheek (Fig. 4a): by Heath,[11] who added a transverse incision below the eye (Fig. 4b) and by Hugier,[12] who also incised the angle of the mouth so as to dissect the whole cheek back to the malar region (Fig. 4c).

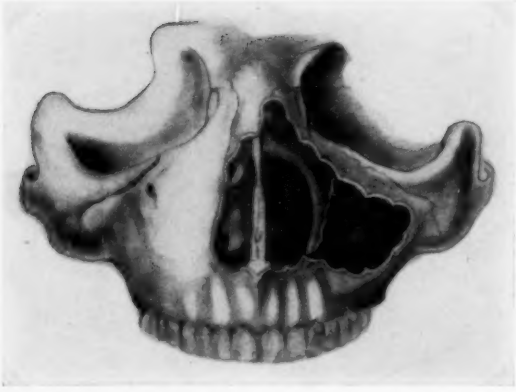

Fig. 5.—The amount of bone removed in Denker's extended operation.

The most extensive and deforming operation of this type was that described by Cheever,[13] who resected portions of both upper jaws by dividing each maxilla from the tuberosity to the middle meatus followed by forcible displacement of the nasal septum downwards (Fig. 4d).

The only useful sublabial operation is Denker's extended operation[14] with removal of the anterior wall of the maxillary antrum, the antronasal wall and part of the frontal processes of the maxilla and nasal bones.

These operations are on the whole more satisfactory than those going directly through the nose but have the same disadvantage that the line of approach is directed towards the sphenoidal area rather than towards the nasopharynx.

Looking again at a sagittal section through the nose it would appear that an approach through the mouth and palate would offer the most direct access. This approach has appealed to surgeons for some years and Hanau Loeb in his classical textbook on operative surgery of the nose, throat and ear (1917)[15] remarks, "Tumors involving the posterior portion of the nose and nasopharynx may be reached by splitting the soft palate and resecting the hard palate to the extent necessary." Actually his description in the text is of a midline incision

from behind the incisor teeth anteriorly dividing the mucosa of the hard palate and the whole thickness of the soft palate as far as the base of the uvula.

Blair,[16] in 1931, used an incision of this type in dealing with a case of choanal atresia.

Several other methods of splitting the soft palate in a more or less longitudinal direction have also been described but none of them is in any way comparable in efficiency of exposure to that favored by Loeb. Figures 6 and 7 are illustrations taken from his book to illustrate the exposure obtained. The main disadvantage of this method is that retraction of the flap laterally is by no means as easy as is suggested, the thickness of the soft palate is very considerable and the exposure is not as good as appears theoretically possible especially in the fossae of Rosenmuller. In addition, the greater the retraction, the more is the posterior part of the soft palate drawn forwards, thus limiting the exposure of the posterior nasopharyngeal wall still further.

Transverse incision with separation of the hard from the soft palate was curiously enough not considered until 1929 when Precechtel[17] suggested such an approach for the surgical treatment of choanal atresia (Fig. 8a). Several surgeons subsequent to this have advocated modifications of a transverse palatal approach through the mucosa of the hard palate for the same condition; Steinzeug[18] using a curved incision angulated anteriorly just inside the alveolus and containing in it the main vessels (Auerbach's incision for removal of the sphenopalatine ganglion) (Fig. 8b), while Schweckendiek[19] and Neto[20] used an H-shaped incision (Fig. 8c). Ruddy[21] in 1945 described a curved incision similar to that used by Precechtel but lying rather more anteriorly (Fig. 8d) while Owens[22] in 1951 recommended an incision similar to that suggested by Steinzeug but without the anterior angulation (Fig. 8e); an incision similar to that recommended by Dorrance[23] in his 'push back' operation for the repair of cleft palate. All these operations have been designed for the treatment of choanal atresia and it is remarkable that no attention appears to have been given to the value of such an approach for dealing with pathological conditions of the nasopharynx itself.

The author in 1946 used an approach of a somewhat similar nature to that advised by Precechtel and Ruddy to remove a mixed salivary tumor of the nasopharynx growing from the region of the eustachian cushion and had the opportunity of using a similar incision

Fig. 6.—Sagittal mid-line incision through hard and soft palate.

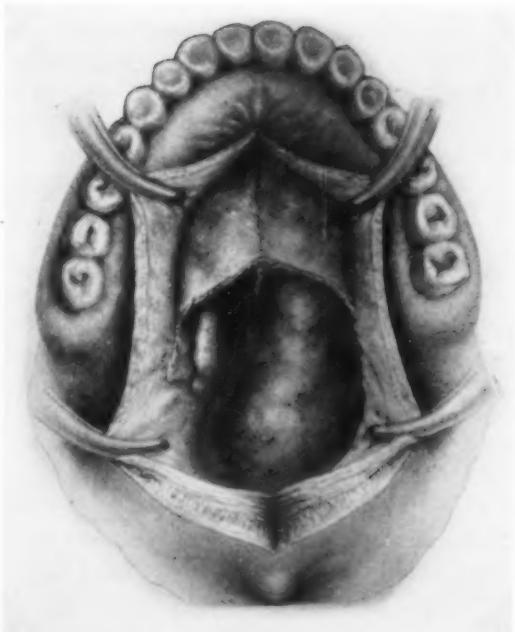

Fig. 7.—Bone has been removed from the posterior part of the hard palate exposing a nasopharyngeal fibroma.

in some ten further cases for varied conditions of the nasopharynx with such satisfactory results that the suggested method employed was described in a communication to the Royal Society of Medicine in 1951.[24]

The details of the method of approach are as follows: The position adopted in young patients is the dorsal recumbent position with a sand bag or pillow under the shoulders and with the head fully extended, the surgeon sitting at the head end of the table in a position similar to that for removal of tonsils under general anesthesia. In adults and particularly in elderly people the neck cannot be sufficiently extended and a more satisfactory position is with a pillow under the neck and shoulders and the head end of the table raised so that the head of the patient is both elevated and thrown back as far as possible. In this case the surgeon stands on the right side of the patient as in most operations on the nose.

The mouth is opened to its fullest extent by means of a Boyle-Davis gag. Anesthesia is carried out with a rubber or polythene intratracheal tube passed through the more suitable side of the nose. If an intranasal tube is likely to prove inconvenient a tube is passed through the mouth. In such a case it should be a latex-armored tube as it will be beneath the tongue plate of the Boyle-Davis gag and must therefore resist pressure. The laryngopharynx is occluded by a gauze pack moistened with saline.

Illumination may be by head mirror or head lamp but illumination from above and behind the surgeon by conventional lighting is usually sufficient.

The palate is infiltrated with novocaine containing a small amount of adrenalin over an area covering the region of the junction of hard and soft palate and extending backwards on each side along the pterygomandibular raphe.

The exact position of the posterior border of the hard palate is identified by puncture of the tissues in several places with a long straight needle. An incision, convex forwards, is made from the inner side of one tuberosity to the other so as to be about ½ cm in front of the posterior border of the hard palate (Fig. 9a). This is necessary to allow a reasonable overlap of soft tissue on subsequent suture thus minimizing the possibility of a fistula occurring after operation.

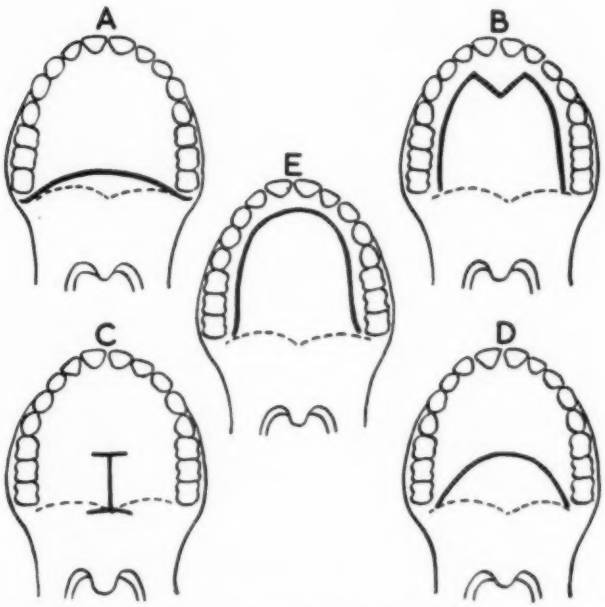

Fig. 8.—Transpalatine incisions for dealing with choanal atresia. A. Precechtel. B. Steinzeug. C. Schweckendiek; Neto. D. Ruddy. E. Owens.

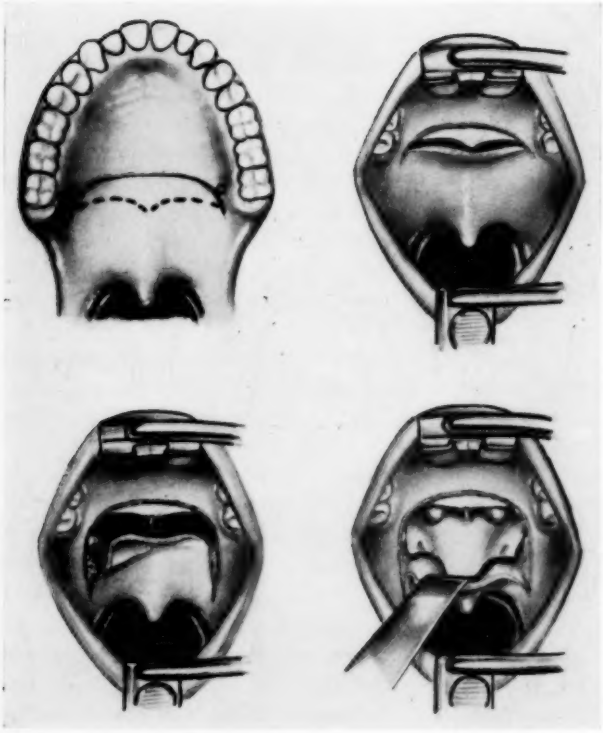

Fig. 9.—Stages in operation for transpalatine approach to the nasopharynx. A. Incision. B. The nasopharynx opened by division of the upper surface of the soft palate. C. Incision extended laterally along pterygomandibular raphe exposing tendon of tensor palati muscle and hamulus of internal pterygoid plate. D. Soft palate retracted exposing the whole of the nasopharynx.

Laterally the incision is posteromedial to the palatine foramina so that only small posterior branches of the vessels are divided. The incision is made with a small-bladed, long-handled knife and diathermy should not be used; it is unnecessary and delays healing.

The incision goes right down to the bone of the hard palate and the mucosa is separated backwards with a sharp raspatory as far as the posterior edge of the bone. Minor difficulty in separation may be encountered with the transverse ridge of the palate and the rather prominent median spine. The mucosa of the upper surface is incised at the posterior border of the bone and the nasopharynx is thus opened (Fig. 9b). At this stage it is convenient to remove the projecting spine with a gouge. The incision is carried laterally and backwards until the pterygomandibular raphe is reached and passes backwards along the raphe for a distance of about 1 cm. In the depth of this part of the incision the hamulus of the internal pterygoid plate is identified with the tip of the finger (Fig. 9c). It is divided with a small gouge or alternatively the tendon of the tensor palati muscle passing across it may be divided. Division of the tendon on both sides appears to result in no subsequent functional disability. No other structures of any importance are encountered by an incision in this plane.

The mucosa of the upper surface is now further divided laterally passing along the anterior border of the eustachian tube. The raw surface exposed gets progressively wider in its lateral parts and the upper incision does not extend as far as the lower.

The soft palate is now quite mobile and can be pushed downwards and backwards for a considerable distance exposing the whole of the nasopharynx (Fig. 9d); a retractor may be used but it is rarely necessary if the incision has been made sufficiently long. Both eustachian tubes, the fossae of Rosenmuller, the posterior border of the nasal septum and the posterior ends of the inferior turbinates can be easily identified.

The main indications for this exposure of the nasopharynx are for the removal of simple tumors and for purposes of exploration and biopsy. This latter is done in cases in which a malignant tumor is diagnosed clinically but histological examination of the specimen removed by other means has been inconclusive. It is also of the very greatest value in cases of that type of submucous carcinoma of the fossa of Rosenmuller in which there is no evidence of neoplasm

on clinical examination of the nasopharynx but the symptoms suggest its presence, e.g., unilateral maxillary nerve pain combined with abducent palsy or with unilateral ptosis or diminution of lachrymal secretion without evidence of any other pathological conditions likely to produce a lesion of the sympathetic nerve.

In such cases a vertical slice of tissue should be removed from the whole thickness of the mucous membrane of the posterior wall of the fossa of Rosenmuller 2 cm in length and ½ cm wide.

The author has had five cases in which a positive histological diagnosis of carcinoma has been obtained in the absence of any visible sign of tumor in the mucous membrane, even on exploration.

Bleeding is of a minor nature and the wound is easily sutured as the soft palate comes naturally back into position. An attempt may be made to suture the upper surface but sutures tend to cut out of the mucosa of the upper surface of the hard palate and their use does not seem to be of much value.

Healing is rapid and there is very little postoperative discomfort; no treatment is necessary other than a simple mouth wash, although an antibiotic may be used. In some cases it has been necessary to perform this operation a second time, in one case five years later; healing in all these second operations has been uneventful.

The foregoing operative procedure was employed in several cases for the removal of a residual malignant growth after a full course of radiotherapy had been given, but subsequent experience and the considerations governing the treatment of malignant growths of the nasopharynx in general, have materially modified one's view of the most desirable procedure in such cases.

It must be accepted that in the present stage of our knowledge and resources the only method of treatment of malignant disease of the nasopharynx which holds out hope of cure is the use of some form of radiotherapy, whether by conventional x-rays, convergent beam therapy, radium or some form of super voltage therapy.

The disadvantages of treatment by these means are manifest. In the first place there is the difficulty of early diagnosis which has already been stressed and also the difficulty in estimating accurately the size and extent of the primary growth by conventional methods of exam-

ination. These disadvantages can be overcome by the simple exploration just described.

During treatment there is difficulty in checking the accuracy of direction of beam and measurement of dosage, while there is also need to watch the clinical effect of the radiation on the tumor during the course of treatment. After treatment has been completed it is desirable to have easy access to the area of the primary growth if local supplementary treatment needs to be carried out, e.g., insertion of irradiated gold grains, diathermy coagulation, etc., and finally it is of vital importance to provide ease of satisfactory routine examination of the area of the primary growth during follow-up.

To overcome these difficulties and to satisfy these needs surgical intervention is essential, and on this account the operation of palatal fenestration has been devised.

PALATAL FENESTRATION

Preparation for operation and preliminary steps are the same as for the transpalatine exploration but a light obturator without teeth is made before operation extending backwards to about 2 cm behind the edge of the hard palate.

Anesthesia is invariably by intubation through the mouth.

Incision. A transverse incision is made slightly convex forwards from the posterior edge of one tuberosity to the other at the level of, or just anterior to, the posterior border of the hard palate; it need not reach the bone (Fig. 10a). The mucous membrane is dissected backwards including some submucous tissue so that the flap is about 1 mm in thickness. After the flap has been dissected backwards for about 1 cm the incision is deepened through the glandular and fatty tissue until the aponeurosis of the soft palate is exposed (Fig. 10b). This glandular and fatty tissue is now separated from the aponeurosis forward to the edge of the bone and is removed (Fig. 10c).

The aponeurosis is divided at the posterior border of the bone and the nasopharynx is thereby opened (Fig. 10d).

The cut edge of the mucosa of the upper surface of the soft palate is now sutured to the cut edge of the lower surface with three catgut sutures (Fig. 11); the removal of the transverse strip of glandular tissue has facilitated accurate approximation of the edges. A median

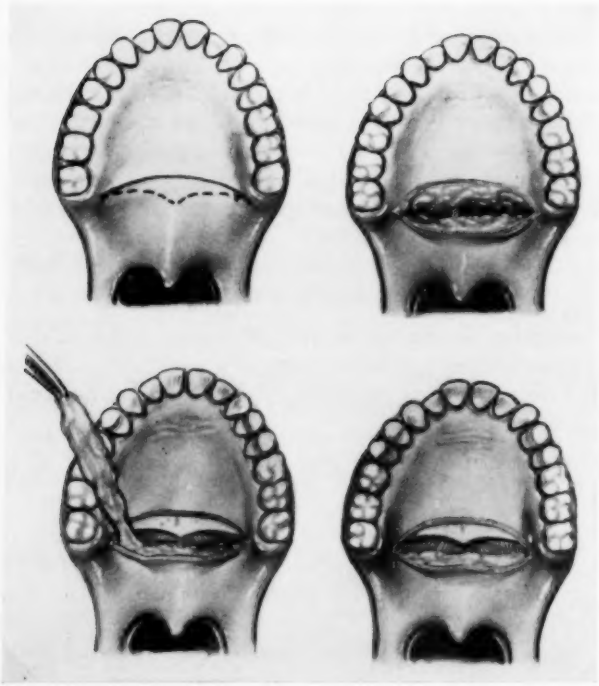

Fig. 10.—Palatal fenestration. A. Initial incision. B. Mucous membrane flap dissected backwards. C. Fatty and glandular tissue removed exposing aponeurosis of palate. D. Nasopharynx opened at posterior border of the hard palate.

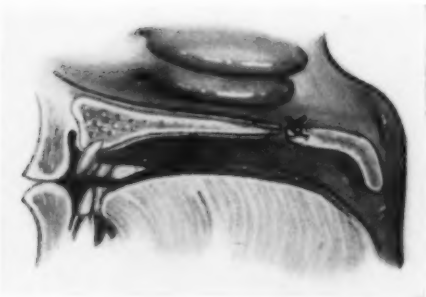

Fig. 11.—Sagittal section showing suture of upper and lower surfaces of front edge of soft palate.

incision is now made as far forwards as is convenient and triangular flaps are outlined (Fig. 12a) and dissected from the bone as far outwards and backwards as the vascular pedicle of the posterior palatine foramina. The pedicles are coagulated and the flaps removed (Fig. 12b). The bone of the hard palate is divided along the periphery of the exposed area with a mallet and long-handled curved gouge, care being taken to avoid damage to the underlying mucous membrane of the nasal floor. It is usually possible to remove the whole of the freed area of bone by levering it up from the middle line anteriorly without damage to the mucous membrane, the thin bone of the nasal septum fracturing quite easily (Fig. 12c). Sharp edges of bone at the periphery can be nibbled away with a sphenoidal punch forceps or removed with a gouge and mallet. The mucous membrane of the posterior border of the septum is divided and a submucous resection of the posterior part of the nasal septum is now carried out from below as far upwards as the alae of the vomer which are left intact (Fig. 12d).

The mucous membrane of the septum on both sides is divided from behind using a pair of long fine curved scissors starting posteriorly at the base of the vomer and curving forwards to reach the palate at the anterior margin of the area of bone removed (Fig. 13a).

This incision is now carried out laterally on each side so that two flaps of mucous membrane are formed each with its base at the lateral margin of the bony removal and consisting of the floor of the posterior part of the nose with a triangular area of the posterior

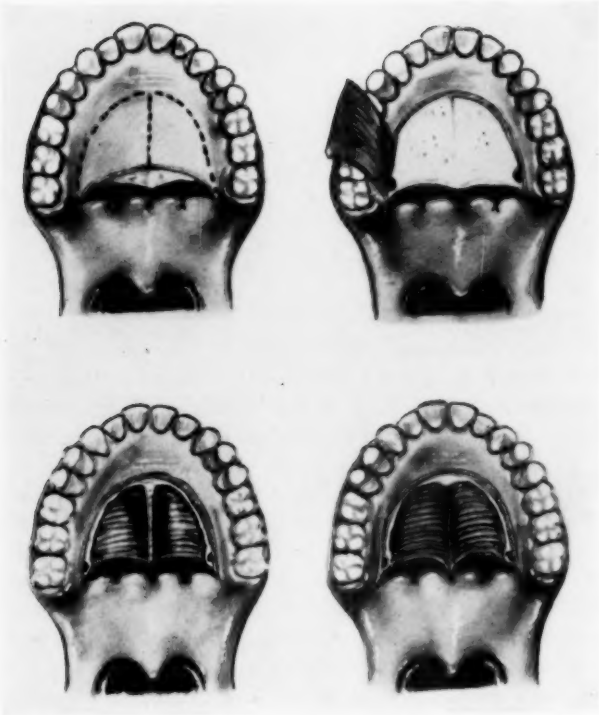

Fig. 12.—Palatal fenestration continued. A. Incision for palatine mucosal flaps. B. Flaps are removed and vascular pedicles coagulated. C. Bone of hard palate has been removed. D. Submucous resection of posterior part of septum carried out from below.

part of the septal mucosa attached (Fig. 13b). These flaps are sutured to the edge of the mucosa of the lateral part of the hard palate so as to cover all raw surfaces on the lateral walls (Fig. 13c).

Any redundant mucous membrane can be removed but it is important to cover the posterolateral angle of the fenestration.

A view is now obtained of the vault and posterior wall of the nasopharynx including the eustachian orifices and the fossae of Rosenmuller (Fig. 13d). In the anterior part the inferior turbinates are seen and the basal part of the septum at the base of the skull. Portions of the posterior ends of the inferior turbinates may be removed if it is considered necessary. There is very little oozing of blood as all raw surfaces should be covered. All blood clot is sucked away from the nasal cavities and from the cavity behind the soft palate and the obturator is inserted. There are no likely postoperative complications and no dressing is required, but an antibiotic is given for a week.

Healing is rapid and if all raw surfaces have been covered should be complete within fourteen days.

The obturator is removed and the mouth washed out after each meal, while the nose and postnasal cavity are irrigated with a salt and bicarbonate solution night and morning.

This operation is usually carried out before radiotherapy treatment is instituted. If a definite diagnosis has not previously been made, a simple transpalatine exploration is carried out and the fenestration is not undertaken until the histological diagnosis has been established.

If there is doubt as to whether the area of operation will involve the primary growth, operation is deferred until about 1000 r of radiation has been given and treatment is then suspended for a fortnight after operation. If, however, the growth is of a diffuse nature such as a lymphosarcoma or involves the posterior part of the nasal septum or the upper surface of the palate then operation is not carried out until about six weeks after the course of treatment has been completed. A temporary obturator is used until tissues have settled down to their permanent shape. There is very little disability from such a procedure and the ultimate discomfort is of no greater order than is involved in the wearing of an upper denture. Many patients, of

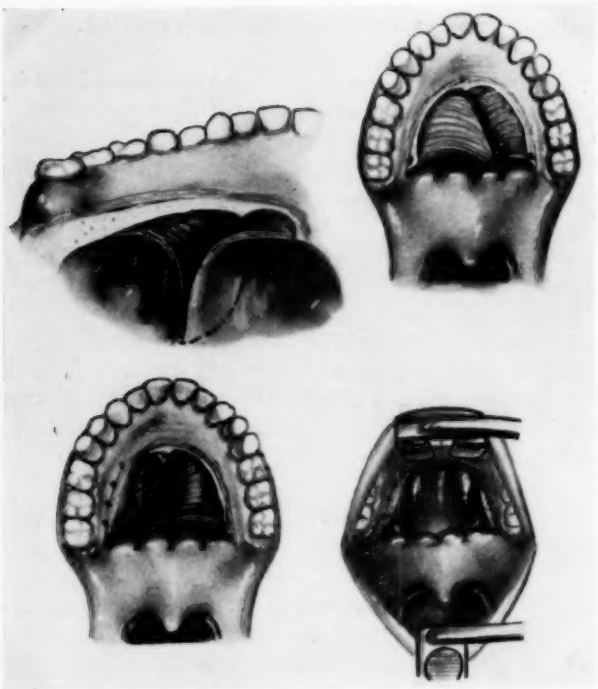

Fig. 13.—Palatal fenestration continued. A. Line of incision for division of septal mucosa. B. Mucosal flap of floor of nose and back of nasal septum has been raised. C. Flap on right side has been sutured to the edge of mucosa of hard palate. D. View of nasopharynx at completion after both flaps have been sutured.

course, already have such a denture and a new obturator may not be necessary.

NASOPHARYNGEAL FIBROMA

Tumors of this type may be dealt with satisfactorily in those cases where the growth is of a semipedunculated nature or is of a comparatively avascular type, by means of the transpalatine approach, but in those more common cases in which the tumor is of an intensely vascular nature many difficulties may arise in an attempted primary excision.

The tumor is associated with large venous channels so closely associated with its capsule and embedded in its fibrous tissue stroma that they cannot easily be picked up and clamped or tied, while diathermy coagulation presents considerable difficulties in the presence of very free bleeding; even comparatively small vessels show no tendency to contract or seal themselves off, although coagulation is of course much easier than with large venous spaces.

Quite apart from the tumor itself there is general dilatation and hypertrophy of the external carotid arterial system and particularly of the internal maxillary and ascending pharyngeal branches, so that even the simple palatine incision made to provide access may be a distressingly vascular procedure; furthermore, the tumor may be in contact with the upper surface of the palate and cause pressure atrophy of the posterior part of the hard palate so that it contains no bone and the initial incision designed to expose the surface of the bone of the hard palate goes directly through the thin attenuated palate and the tumor itself is incised.

The resulting bleeding, which responds very poorly to the use of adrenalin or thrombin, or even a coagulating diathermy current can be extremely disconcerting and firm pressure may need to be applied for some considerable time before any further progress can be made. It may even be that any attempt at removal has to be deferred for some days during which blood transfusions are given, and the subsequent interference may be approached in too half-hearted a way to insure success. Such a case will rarely be discussed by the surgeon and almost certainly never reported.

On the other hand, should the tumor be of a comparatively avascular type, it may be excised and the base coagulated. Even though the tumor may recur within a few months the surgeon will be encouraged to tackle a subsequent case, which may be very vascular, in a more optimistic mood than is justified. Unfortunately, such is human nature, his recital of his experiences is likely to dwell more on his comparatively successful rather than on his unsuccessful efforts.

Radiation therapy has been used for many years in an effort to overcome the disadvantages of surgical intervention and in general it has seemed that conventional x-ray treatment has not been of much value, although insertion of radium needles has provided that additional fibrosis which has enabled supplementary surgical procedures to be less hazardous. Treatment by supervoltage therapy seems to

produce results comparable with those produced by radium so that it would appear that if radiation is to be used in combination with surgical treatment it should be by radium or supervoltage therapy rather than by conventional x-rays.

Whereas with malignant growths it is suggested that treatment is primarily radiotherapeutic and surgical intervention is ancillary, with nasopharyngeal fibromata surgical extirpation takes a more prominent place in treatment and radiotherapy can be looked upon as a means of enabling surgical removal to be carried out in cases which would otherwise entail a very grave risk.

Our knowledge of the pathology of these tumors is still very incomplete and some of them show quite definite invasive tendencies, entirely different from the bony atrophic pressure effects produced by the majority. They seem to come into the class of fibrosarcomata rather than simple fibromata and in such cases radiotherapy will assume a more prominent role in treatment.

In view of these considerations the technique of treatment now being carried out at the Middlesex Hospital, London, is to treat the patient with a course of radiation using supervoltage therapy before any surgical treatment is attempted. Some six weeks after the course of treatment has been completed a fenestration operation is performed, the growth is excised and the base is coagulated. The lateral parts of the incision may need to be extended as in the transpalatine exploration operation to enable the tumor to be more easily dealt with. It is now possible to inspect the site of the tumor at regular intervals and to repeat electrocoagulation if at any time there appears to be a recurrence of the growth. An additional advantage is that in the event of any subsequent bleeding it is a very simple matter to pack the nasopharyngeal cavity through the fenestrum; two patients have carried out this proceeding themselves using a small sterilized turkey sponge kept at hand in a glass jar. Although fenestration is not generally carried out until six weeks after the course of radiation, it may be advisable to do it during the course of radiation if recurrent hemorrhage of sufficient magnitude to cause alarm should occur. In such a case no attempt is made to remove the growth at the time of operation, but the fenestration makes control of bleeding during treatment much easier as pressure can be applied, or the bleeding surface coagulated under local anesthesia very promptly and simply if it is necessary. No attempt at extirpation is made until six weeks after radiotherapy has been completed.

With adoption of the routine described, treatment of these distressing and alarming growths has been much simplified and the results of treatment have been extremely satisfactory. The patient tolerates the obturator very well and there is no apparent disability or deleterious effect on growth or development of the alveolus or palate. As the majority of patients are boys, a new obturator may need to be made every twelve months or so because of the increase in size of the alveolus and palate during the ensuing few years. The main postoperative discomfort is associated with dry crusting of the vault of the nasopharynx secondary to the radiation, but this is easily dealt with by simple toilet if the patient is of reasonable intelligence. Unfortunately, some of these patients are of rather lower mental standards than average and may need nursing supervision to insure adequate hygiene.

An objection may be raised that a permanent fenestration is a high price to pay for treatment of a condition that may be dealt with in some cases by less radical measures and which may naturally retrogress after some years. The answer to such an objection is that this condition is one of undoubted gravity: many cases, probably many more than is generally admitted, are fatal and the line of treatment suggested, although more drastic than is necessary in some cases, has produced a marked overall improvement in results.

TREATMENT OF CHOANAL ATRESIA

The treatment of choanal atresia provides a special problem of its own. In some cases of bilateral choanal atresia in infants the problem is of an urgent and life-saving character designed to allow adequate nutrition in a very ill and dehydrated patient, and only the simplest measures directed to this end should be considered.

In unilateral cases and in bilateral cases where the urgency of the symptoms is not marked or has been overcome by temporary measures, a planned surgical approach through the palate is the most satisfactory line of treatment.

Two quite distinct problems are present. Firstly, the method of approach and, secondly, the method of dealing with the atresia.

The Method of Approach. Most authors now favor some form of curved transverse incision through the mucous membrane of the hard palate. Such incisions can be divided into those lateral to the area of mucosa supplied by the main palatine vessels and those medial or posteromedial to the vessels.

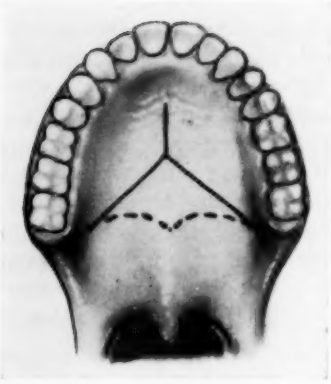

Fig. 14.—Choanal atresia. Line of incision in palate.

Of the former group the incisions of Steinzeug, Owens and Dorrance have already been mentioned; they have the advantage of preserving the blood supply of the mucosal flap but are not ideal for adequate exposure of the posterior border of the hard palate. Nevertheless they have the advantage that the line of incision is far removed from the area of bone removed and a subsequent fistula is not a possible complication. An incision of this type is probably the best in infants because of the poor development of the hard palate and constant crying produces traction on a more posterior incision.

Incisions medial to the vessels such as described by Precechtel and Ruddy have the advantage of being much nearer the area of operation and enable any variety of atresia to be dealt with adequately. There is very little likelihood of a subsequent fistula occurring although it is a more distinct possibility than with a juxta-alveolar incision.

Ruddy's incision is from this point of view more satisfactory than that of Precechtel. The author favors a tripartite incision similar to that of Ruddy but not quite so far forwards, with a midline extension forwards to give greater flexibility of exploration if it should prove necessary (Fig. 14). The actual incision is not of major importance and is a matter that will depend on the preferences of the individual surgeon in any particular case.

The Method of Dealing with the Atresia. The essential points in this respect are a) that enough tissue (bone or fibrous tissue) is

removed so that the resulting choanal orifices should be of adequate size; b) that all raw surfaces of bone should be as far as possible covered by mucous membrane. All surgeons who have considered this subject carefully are in agreement that in the vast majority of cases it is necessary to resect the posterior part of the bony septum. A congenital atresia is rarely a simple fibrous or bony partition: the whole of the choanal region is contracted; the posterior part of the septum is deviated to the side of the atresia, the lateral wall is nearer the middle line, while the hard palate is often more arched; in other words, the whole of the choanal area is smaller than normal rather than merely obstructed.

It must be realized that it is not possible to widen the area to any extent in a lateral direction without interfering with the nerves and vessels of the palatine canal so that the only way of enlarging the area of the choana is to resect part of the posterior border of the nasal septum, thus making the plane of the new orifice oblique; the choanal orifice on the unaffected side will be to some extent larger than previously. In order to preserve the mucous membrane areas which will subsequently be used for covering raw surfaces of bone, the removal of the bony mass forming the atresia and the posterior part of the nasal septum must be done with great care. This will involve removal of part of the bone of the posterior end of the hard palate on the affected side of sufficient extent to expose the mucosa of the nose anterior to the atretic area and removal of a smaller though similar area of hard palate on the unaffected side so as to allow ease of removal of the posterior part on the bony nasal septum. This is the area of removal of bone advised by Ruddy in his description in 1945 of a case on which he had operated.

When as much bone as is considered adequate has been removed, it remains to cover all raw surfaces with mucous membrane. There are four such raw areas involved—roof, floor, lateral wall and septal edge. The last named is the least important as it is only a sharp edge and, provided sufficient bone has been removed to allow the edge to be covered on each side by the remaining septal mucosa, nothing further is necessary.

Precechtel removed no bone anterior to the atresia and no bone from the unaffected side of the hard palate; his incision is rather far back so that his approach is from behind and by means of a vertical incision in the middle of the mucosa of the posterior surface of the atresia he reflects flaps medially and laterally and then, having removed

the bony obstruction, makes similar flaps in the anterior surface, finally suturing the anterior and posterior flaps to each other medially and laterally (Fig. 15).

By this method the lateral wall is covered by mucous membrane and also the posterior border of the septum but raw surfaces are left in roof and floor. His suggestion that the floor of the atresia is covered by suturing the mucosa of the upper surface of the soft palate to that of the floor of the nose anterior to the atresia is optimistic rather than effectual, as they are separated by the whole thickness of the atresia.

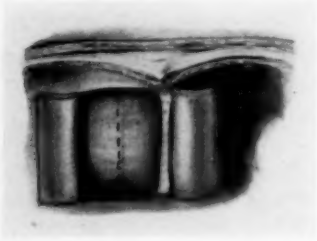

Fig. 15.—Precechtel's method of dealing with the mucous membrane in choanal atresia.

Ruddy did not make any attempt at special preservation of mucosa, nor does he report upon how the nasal cavity anterior to the atresia was opened; presumably the mucous membrane covering both anterior and posterior surfaces was completely removed with the bony mass and reliance was placed on distension with a rubber balloon to keep the orifice open.

Most of the hard palate was removed by Steinzeug and Schweckdiek on the unaffected side, while Owens removes more or less the same amount of bone as advised by Ruddy. He divides the mucosa of the posterior border of the vomer to make submucous resection of the posterior part of the septum easier. He has found this amount of bony removal necessary and satisfactory in several cases up to the time of his paper (1951) and the author is in complete agreement with this (Fig. 16).

The Covering of All Raw Surfaces. It has already been suggested that there are three raw areas to be covered: roof, floor and lateral wall;

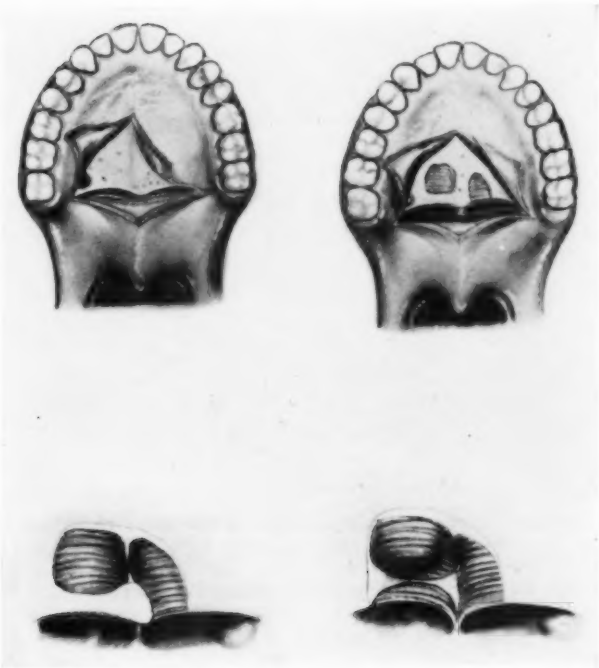

Fig. 16.—Choanal atresia. Steps in removal of bone of palate and septum.

there are also three areas of mucous membrane available for this purpose:

1) The mucosa covering the posterior surface of the atresia.

2) The mucous membrane which originally covered the posterior part of the septum on the unaffected side.

3) The mucosa covering the anterior surface of the atresia.

Owens suggested using this septal flap to cover the roof but leaves the lateral wall and floor. He treats patients after operation by removing crusts from the raw surfaces and passing suitable dilators.

Blair, Steinzeug and Schweckdiek use the mucosa of the posterior part of the septum on both sides to cover the raw base of the vomer

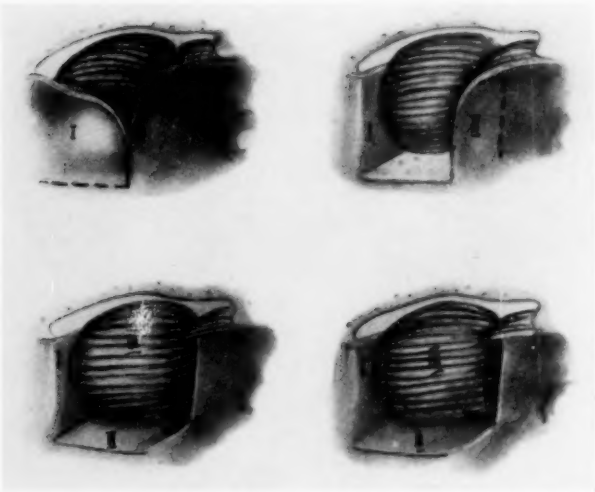

Fig. 17.—Choanal area viewed from behind after removal of bone. A. Line of incision in posterior aspect of atresia (area 1). B. Area 1 covers the lateral wall; line of incision in septal flap (area 2). C. Area 2 covers roof of choana. D. Line of incision in anterior aspect of atresia (area 3).

in the roof of the nose. This would seem to be the least important part to be covered as it is disposed in an anteroposterior direction and fibrous contraction of this would not be likely to contract the new choanal orifice. The following procedure is suggested as a simple means of procuring complete covering of all raw surfaces and has proved successful in obviating the need for any postoperative treatment or dilatation.

To insure success it is desirable that the mucous membrane of both surfaces of the atresia should be uninjured during the removal of the bony mass, hence the importance of adequate removal of bone from the hard palate to enable the posterior part of the septum and the bony mass to be removed from below without damage to the mucosa.

The mucosa covering the posterior surface of the atresia (area 1) is designed to cover the raw area of the lateral wall of the choana. Its medial and palatal edges are already free so that it only needs divi-

sion at its upper margin to enable it to swing forwards, its lateral border acting as a hinge (Fig. 17a).

The mucosa of the posterior part of the nasal septum on the unaffected side (area 2) is used to cover the raw surface of the roof of the choana. The flap is formed by dividing the lower palatal edge in the midline and then the anterior edge at the new posterior border of the septum; its upper edge will act as a hinge (Fig. 17b).

The mucosa covering the anterior surface of the choana (area 3) is still intact, it is designed to cover the floor of the nose and will swing backwards on its palatal border; it will need to be divided laterally, above and medially (Fig. 17c). The raw surfaces of the roof and lateral wall are painted with a thrombin solution and the two mucosal flaps are placed in position. A thin-walled rubber tube is now inserted into the nose, its anterior end lying just inside the nostril and its posterior end projecting into the nasopharynx for about 1 cm. It is intended to keep the flaps in position without exerting any considerable degree of pressure (Fig. 17d). Flap three is now laid on the surface of the tube and is sutured to the medial edge of the mucosa of the floor of the nose on the unaffected side: any excess of mucous membrane can be trimmed away (Fig. 18a). The soft palate is now replaced, the mucosa of the upper surface of the soft palate is stitched on both sides to the mucous membrane; on the unaffected side to the mucous membrane from which it was originally divided and on the affected side to the new posterior border of the nasal floor which was originally the upper edge of the mucosa forming the anterior wall of the atresia (Fig. 18b). The original incision in the palate is now sutured; there should be no risk of a fistula developing as the incision overlies intact mucous membrane throughout (Fig. 18c).

The rubber tube can be removed after 24 hours and no special after treatment is necessary. An antibiotic may be given and saline drops instilled into the nostrils every few hours to aid removal of secretions.

It will not, of course, be possible to carry out this rather elaborate procedure if the atresia is of a simple fibrous nature. In such a case the fibrous partition should first be excised and the mucosa of the edges reflected forwards and backwards around the whole circumference. Sufficient bone is removed to enlarge the choana as much as is considered necessary and the anterior and posterior edges of the mucous

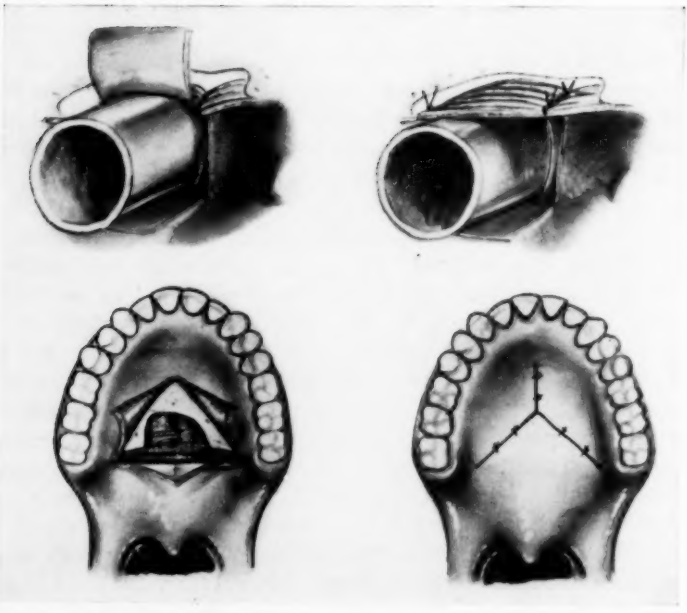

Fig. 18.—A. Rubber tube inserted into nose and projection into naso-pharynx. B. Area 3 sutured at lateral corner and in middle line. C. Upper surface of mucous membrane of soft palate sutured to posterior border of floor of nose. D. Completion of suture of incision.

membrane are then sutured to each other over the raw surface the whole way round. Unfortunately the resulting cicatrix is of a circular nature and will inevitably tend to contract and may need subsequent dilatation. It is for this reason that a similar procedure is not advised in the case of bony atresia.

In cases of bilateral atresia there will only be two areas of mucous membrane available on each side and it is inevitable that a transverse raw strip will be left on the roof immediately behind the new posterior border of the septum, as there is no septal mucosa available to cover it. Provided enough bone of the posterior part of the septum has been removed to insure that the choanal orifices are of sufficient size, there should be no resulting disability as secretions would not be held up even if a transverse cicatricial web were formed in the vault. If it was felt, however, that this was likely to happen it might be desirable

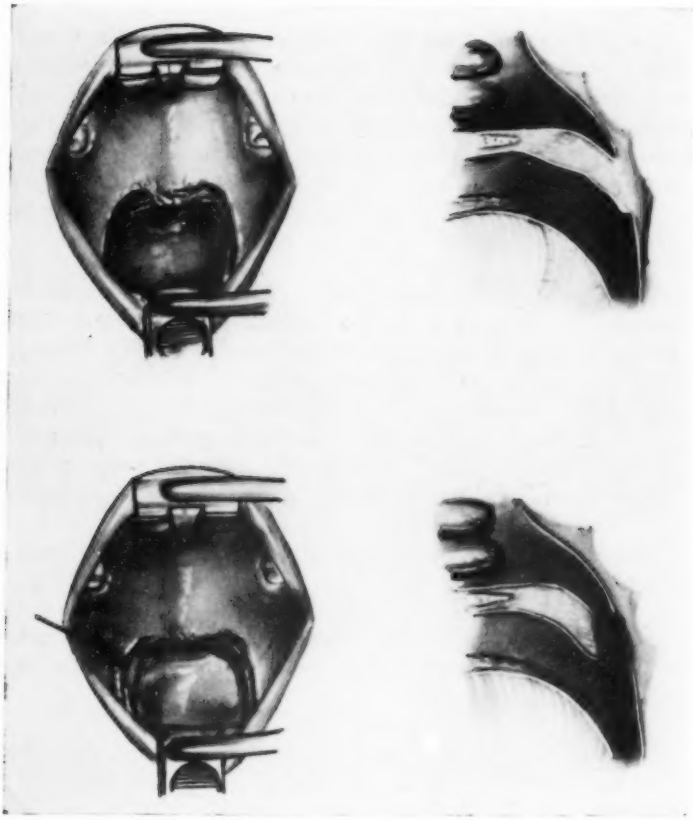

Fig. 19.—A. Stenosis of nasopharynx with adhesion of posterior part of soft palate; faucial pillars to posterior wall of pharynx. B. Sagittal section. C. Incision through scar tissue to separate soft palate from posterior wall. D. Incision is extended backwards on each side and palatopharyngeus muscle is exposed.

to cover the raw surface with a skin graft which could be stuck with thrombin and also sutured at its edges. The author has not had the opportunity of performing this procedure.

STENOSIS OF THE NASOPHARYNX

Surgical treatment for this condition must follow the same rules as apply to choanal atresia, i.e., adequate removal of the tissue producing the obstruction (in these cases scar fibrous tissue) and the covering of all raw surfaces with an epithelial covering; as there is no mucous membrane available, a split thickness skin graft must be used. The extent of the surgical interference will naturally vary with the severity of the case but the same principles must be followed in all cases.

In the most simple type where the stenosis affects only the lower part of the nasopharynx so that the free edge of the velum is adherent in whole or in part to the posterior pharyngeal wall (Fig. 19a, b), an incision is made with a knife through the scar adhesions, separating the muscles of the soft palate from the posterior wall of the pharynx; this incision should pass laterally as far as the posterior pillar of the fauces and then downwards on each side along the posterior border of the pillar until the incision is well below the limit of the scarring.

The incision is deepened in its lateral angle so as to expose the palatopharyngeus muscle and the scar tissue is dissected from its surface (Fig. 19c, d). This freeing of the lateral part of the muscles enables the soft palate to be drawn forwards and the scar tissue removal from the upper surface of the soft palate is continued as far as the middle line from each side.

The upper and lower edges of mucous membrane on the posterior wall of the pharynx are dissected free for about 2 mm from the line of the raw surface. A strip of skin about 6 inches in length and an inch wide is now cut from the thigh and one end is sutured in the middle line of the posterior pharyngeal wall to the mucous membrane above and below (Fig. 20a). It is next sutured in the lateral angle of the incision on one side making sure it is tucked laterally, well behind the palatopharyngeus muscle. The lower suture is easy and is sewn to the lower outer edge of the incision; the upper suture is often difficult and a middle, through and through suture should be inserted to hold the graft to the tissues behind the muscle. The needle should be inserted into the mucous membrane lateral to the anterior pillar of the fauces and passed through the tissues lateral to

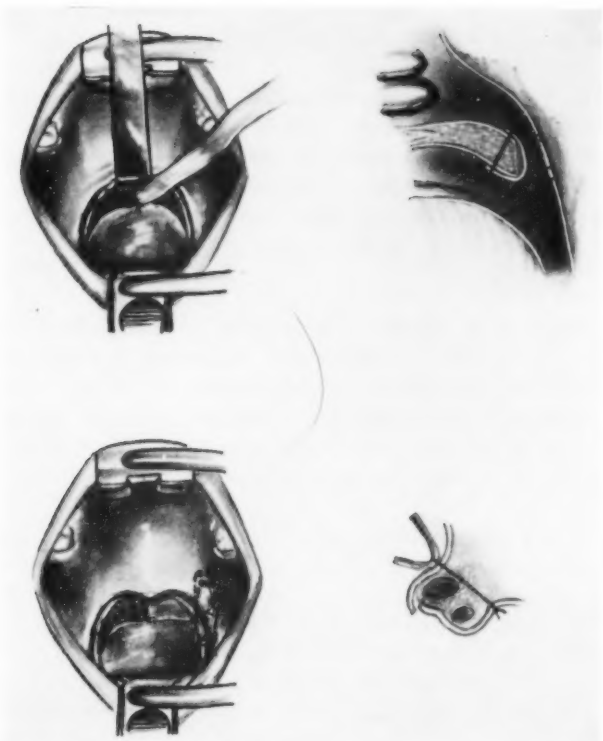

Fig. 20.—A. Skin graft sutured in middle line to posterior wall of nasopharynx. B. Sagittal section of soft palate to show sutures holding graft in position. C. Split tube sutured behind palatopharyngeus to keep graft in contact with lateral angle. For clarity only the posterior part of the graft on the left side is shown. D. Section through tube to show suture.

the tonsillar fossa and posterior pillar; it picks up the skin graft and emerges in similar fashion.

The graft is now brought around to envelop the free edge of the soft palate and held in position by two or three sutures passing through the whole thickness of the palate and both layers of the graft (Fig. 20b).

The graft is now sutured to the opposite lateral angle of the incision behind the palatopharyngeus muscle and finally brought to the middle line of the posterior pharyngeal wall, thus forming a complete epithelial collar lining and covering the raw surface; any excess length of graft is discarded. The raw surface of the graft is painted with thrombin at every convenient stage before the sutures are inserted.

If there is doubt about the security of the upper stitch at the lateral angles a piece of split rubber tubing about 1 inch in length can be sutured in the lateral angle so as to keep the graft in contact with the lateral wall; it will have sufficient rigidity to exercise slight pressure on the upper part of the graft even though it is sutured only in its lower half (Fig. 20c and d).

In more severe cases there is not only more extensive adhesion of the walls of the nasopharynx but probably also loss of tissue of the edge of the velum including the uvula. The function of the palate depends almost entirely on the integrity of the levator palati and palatopharyngeus muscles and, provided these muscles are intact and functioning, the utility of the palate and nasopharynx can be restored. The adhesions are divided as far as possible as already described. A semicircular incision is now made in the roof of the mouth about 1½ cm in front of the posterior border of the hard palate and the mucous membrane stripped from the bone; the nasopharynx is opened at the posterior border of the hard palate as in the operation for transpalatine exploration of the nasopharynx; the tendon of the tensor palati is divided so that the whole of the soft palate can be pushed back and any remaining adhesions are freed from above together with any further removal of scar tissue that may be necessary, particularly around the eustachian cushions.

A skin graft collar of appropriate size is now sutured into position, the lower stitches being inserted from below the palate and the upper ones through the palatal opening.

The incision in the palate is sutured. If the scarring is of such an extent that it is desirable for the velum to occupy a more posterior position, the incision is closed only in its lateral part and no attempt is made to bring the palatal flap forwards in the middle line; a gap of perhaps a half centimeter is left, the floor of which is the bony hard palate; this can be left to granulate or another small skin graft can be sutured into position. If this procedure is necessary, a small light obturator is made to cover the area to prevent the patient playing with the graft with the tip of the tongue.

There is no need for postoperative dilatation and provided the levator palati and palatopharyngeus muscles have not been interfered with the functional result is perfectly satisfactory.

It would be incorrect to assume that the various operations outlined in the foregoing remarks are the limit of what can be attempted by transpalatine surgery. The whole of the area of the body of the sphenoid is open to exploration including the sphenoidal sinuses and the pituitary gland. In one case the author has removed the floor and lateral walls of the sphenoidal sinuses, exposing the periosteum covering the cavernous sinuses in an attempt to remove the remains of a neoplasm primarily treated by radiotherapy; it is certainly the best avenue of approach for cystic tumors of the base of the skull, although for lesions of the pituitary gland it is doubtful if this is the most satisfactory mode of access.

It is to be hoped that with added experience of this approach, many other surgeons will be able to contribute further to its usefulness.

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II

HUMAN OLFACTORY BULB IN NEUROMYELITIS OPTICA: A STUDY WITH SILVER CARBONATE

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The pathology of the human olfactory bulb in diseases of the nervous system has received little attention. Brunner[1] in 1923 by using Nissl technique, found pathological changes in the olfactory bulbs of apparently normal adults, and described the degeneration of ganglion cells and even "a complete absence" of neurons. Smith[2] in 1942 investigated the ratio of the regression of the number of the olfactory nerve fibers during life.

Developments of new histological methods by del Rio Hortega[3,4] made possible a detailed study of normal,[5,6] as well as pathological, structures of the olfactory bulb.

MATERIAL AND METHODS

The olfactory bulbs used in the present investigation were obtained from the post mortem examination of a seven-year-old girl, A. N. (Lab. No. 15492), who died at the University Hospital after an illness of eight months' duration. The clinical findings were blindness, paralysis of the extremities, incontinence and, in terminal stage, lethargy. On examination two months prior to her death the sense of smell was found to be normal. On gross examination the olfactory bulbs appeared normal. Frozen sections were cut at 15 microns and impregnated with several variants of the silver carbonate technique.

HISTOLOGICAL EXAMINATION

The first layer (fila olfactoria) could not be conclusively examined because of slight damage during the removal, but indications of degeneration of the nerve fibers were found.

The second (glomerular) layer was severely degenerated. The synaptic structures normally formed by the neurites of fila olfactoria

From the Laboratory of Neuropathology, Neuropsychiatric Institute, University of Michigan Hospital, Ann Arbor, Michigan.

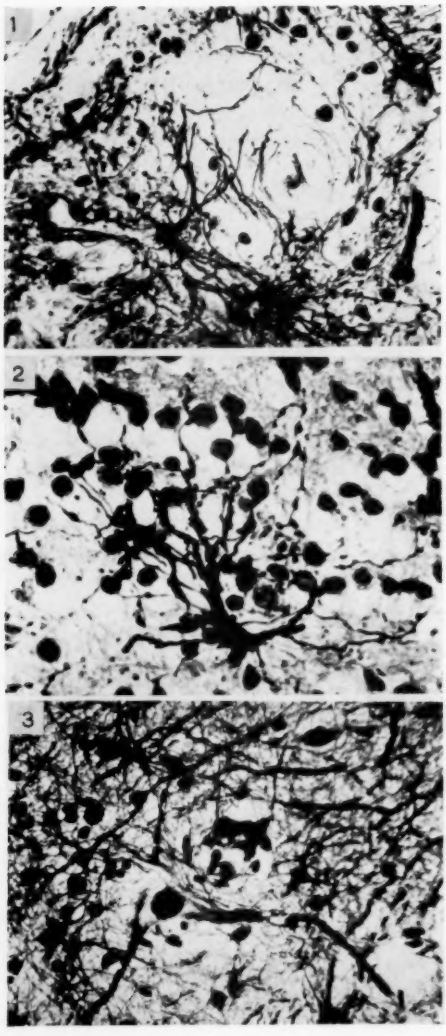

Fig. 1.—Proliferated astroglia around degenerated glomeruli. The glomerular synaptic structures are destroyed and the small intra- and periglomerular neurons degenerated. Photomicrograph. Zeiss Neofluar 40x.

Fig. 2.—Proliferated astrocyte with pearl-like swellings of the processes. (Ganglionic layer close to the glomerular layer). Photomicrograph. Zeiss Neofluar 40x.

Fig. 3.—Mitral cell with a large nucleus, damaged cell body and fragmented dendrites and neurite. Photomicrograph. Zeiss Neofluar 40x.

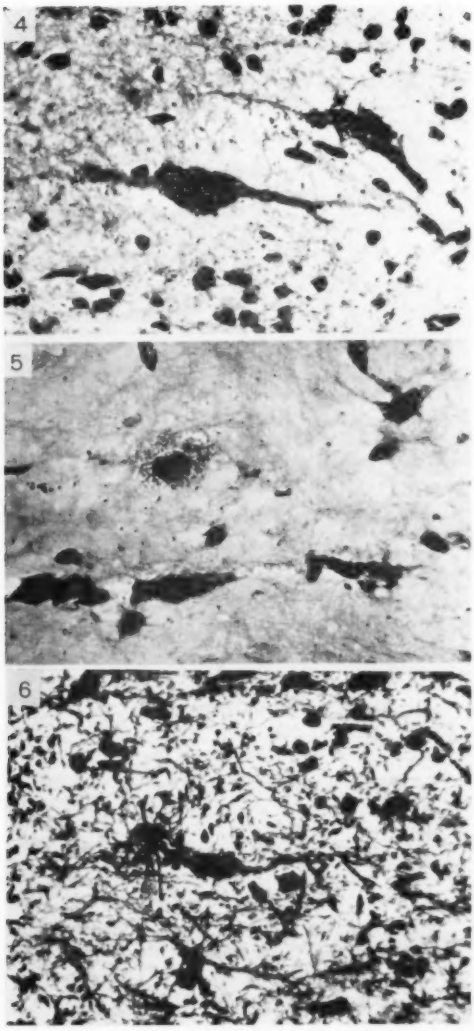

Fig. 4.—Two damaged neurons with cell bodies impregnated homogeneously black and partly broken off processes. Photomicrograph. Zeiss Neofluar 40x.

Fig. 5.—Neuron in advanced stage of disintegration. Only the nucleus is visible, the cell body is granular and neither the cell outlines nor the processes can be seen. Photomicrograph. Zeiss Neofluar 40x.

Fig. 6.—Damaged neurons surrounded by proliferated glial cells. Photomicrograph. Zeiss Neofluar 40x.

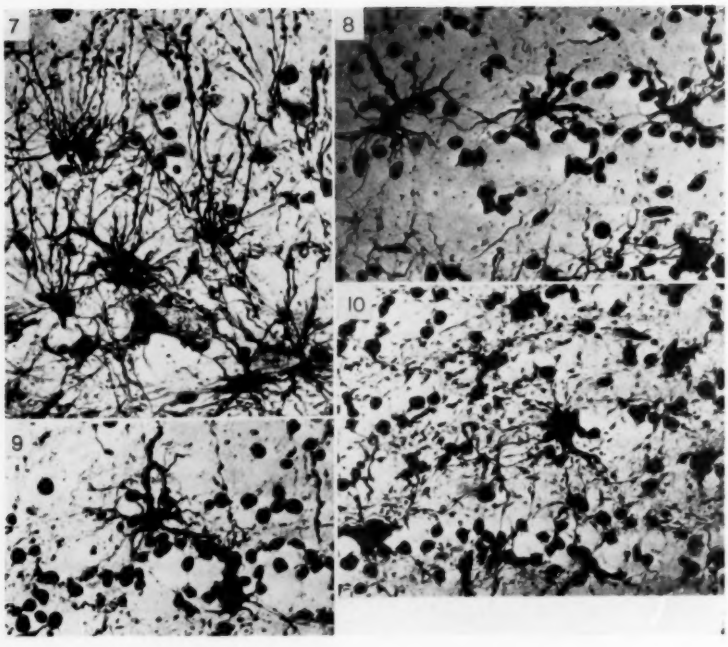

Fig. 7.—Proliferated astroglia with numerous long processes. (Ganglionic layer, close to the layer of the olfactory nerve fibers.) Photomicrograph. Zeiss Neofluar 40x.

Fig. 8.—Linear arrangement of astrocytes; the processes of these elements are thickened. (Borderline between the ganglionic and the olfactory tract fibers layer.) Photomicrograph. Zeiss Neofluar 40x.

Figs. 9 - 10.—Astrocytes from the ganglionic layer: 9: some elements have thickened processes; 10: others have fine and long processes. Photomicrograph. Zeiss Neofluar 40x.

and dendrites of the mitral cells were destroyed. The astrocytes in this layer showed a marked proliferation and replaced the destroyed glomeruli. The intra- and periglomerular neurons, which are numerous in normal bulbs, were rarified, and those remaining showed advanced degeneration (Fig. 1).

In the third (ganglionic) layer different structures showed various degrees of degeneration. The astroglia cells located between the glomerular and the ganglionic layers were proliferated and the processes were thickened and had pearl-like swellings (Fig. 2). The mitral and tufted cells were severely damaged. The body of some of these cells could still be recognized, although the dendrites were swollen and fragmented (Fig. 3), while in more advanced stages of degeneration, disintegrating cell bodies were impregnated homogeneously dark and their processes swollen and fragmented (Fig. 4). In other cells where the destructive process was still more advanced, only the nuclei were present, and the cell outlines could not be seen (Fig. 5). The glial reaction around the severely damaged neurons was very conspicuous. The astroglia were swollen and showed ameboid degeneration (clasmatodendrosis); also, numerous swollen oligodendroglia and microglia cells were present (Fig. 6).

The neurites, which arise from the neurons in the ganglionic layer, were accompanied by proliferated astroglia. The bodies of these astrocytes were large and their processes long and numerous. The majority of the astroglial processes were oriented along the neurites, which in this area ran perpendicularly to the layer of the olfactory tract fibers until they reached it, then they assumed parallel arrangement. These astrocytes, although distinctly proliferated, showed no degenerative changes (Fig. 7). Side by side with areas of astroglial proliferation there were areas with apparent degeneration. In the latter areas the astrocytes were proliferated and lined up forming a demarcation line; these cells had short and thickened processes (Fig. 8). The astroglia located between the small ganglion cells displayed a wide variety of degenerative changes. Some cells had short, thick processes (Fig. 9), while others had long, fine, fibrillary processes (Fig. 10).

In the fourth (olfactory tract fibers) layer the nerve fibers showed degenerative changes such as swellings, and bulbs of degeneration (Cajal[7]) (Fig. 11). The astrocytes had slightly thickened processes and were following the parallel course of the nerve fibers (Fig. 12).

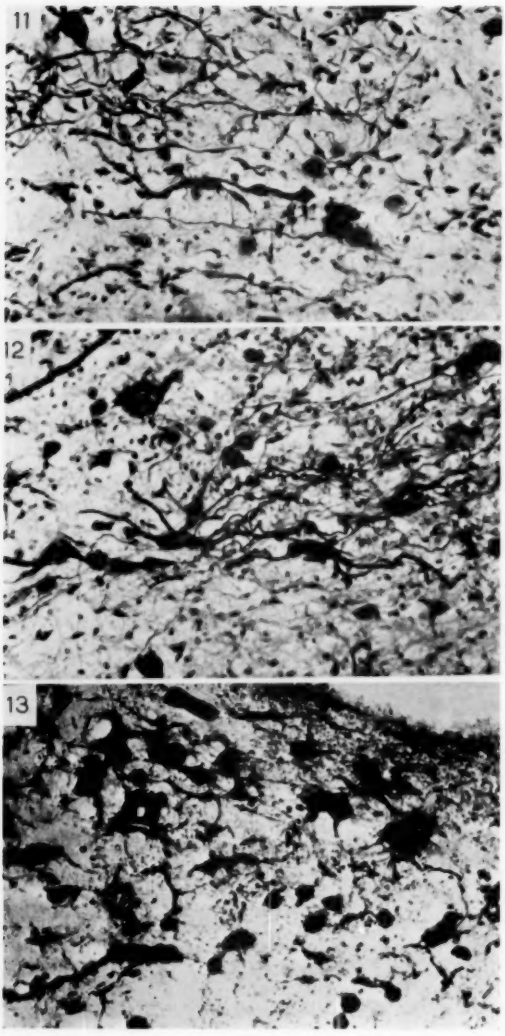

Fig. 11.—Degenerative changes of the nerve fibers. (Layer of the olfactory tract fibers.) Photomicrograph. Zeiss Neofluar 40x.

Fig. 12.—Astrocyte with long, slightly thickened processes. (Layer of the olfactory tract fibers.) Photomicrograph. Zeiss Neofluar 40x.

Fig. 13.—Astro-, oligo- and microglia on the dorsal surface of the olfactory bulb. Photomicrograph. Zeiss Neofluar 40x.

The dorsal surface of the olfactory bulb showed an abundance of astroglia, oligodendroglia, and microglia in various stages of degeneration (Fig. 13).

COMMENT

The changes in the olfactory bulb described here were caused by neuromyelitis optica of eight months' duration. Apparently two months prior to the death of the patient the function of the first cranial nerve appeared to be intact, but because no special studies were made, it is impossible to determine if it was impaired.

The very conspicuous destruction of the glomeruli and the degeneration of the ganglion cells suggest that the patient was anosmic prior to her death. The degeneration of the neurons varied from very slight damage to complete disintegration, but no neurons of normal appearance were present. The layer of the olfactory nerve fibers was also degenerated.

Each of the layers showed a glial response characterized by a) the specific cytoarchitectonic of each layer, and b) the severity of the damage. Around the destroyed glomeruli only glial proliferation was present. In the ganglion cell layer the astroglia cells located close to the neurons showed varying degrees of degenerative changes. The astrocytes which accompany nerve fibers were proliferated and showed very few, or no signs of degeneration.

It is the purpose of this investigation to show the response of the complicated structures of the olfactory bulb to the neuromyelitis optica. This disease, which involves the entire central nervous system and is known to attack the second and the third cranial nerves, is able to cause severe changes in the olfactory bulb, as shown in this case. The glia in the bulbus olfactorius displays the same protective activities toward the damaged ganglion cells and nerve fibers as in the other parts of the nervous system. The changes of the glia are both degenerative and proliferative, a fact which suggests that in this chronic disease both degeneration and regeneration processes are active.

SUMMARY

The pathological changes of the olfactory bulb in neuromyelitis optica follow the general pattern of changes in the central nervous system. They involve both the neurons and the glial elements. The pattern of the pathological process is determined by the cytoarchitec-

ture of the olfactory bulb and is specific for each layer; it is also related to the severity of the damage.

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III

SURGERY OF THE WINDOWS OF THE LABYRINTH IN OTOSCLEROSIS

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At the present time, functional surgery of otosclerosis, which is almost as old as otology itself, is being transformed even more profoundly. It is particularly interesting to note that its history can be traced in three stages expressing a sort of balancing between the direct action on the lesions of the windows and the indirect palliative action at a distance from the focus paralyzing the affected organ.

The first stage begins in 1876 when Kessel practiced functional surgery on the directly affected ear, the oval window and the stapes. In the same way, Urbantschitsch in his thesis, "Disorders of the Ear," speaking of Kessel's operation said: "The experiments carried out on the cadaver prove that it is extremely difficult to detach the base of the stapes from its connections; one must also in the mobilization of this ossicle resort to circumcision, an operation which runs certain risks as it can result in suppuration of the middle ear which might easily spread to the labyrinth."

From 1880 to 1890 nearly all treatises on this special branch of pathology mention this kind of operation in cases of ankylosis of the stapes. Schwartz, Gelle, Boucheron, Moure and many other otologists of this epoch performed this operation with good functional results. In this way, Miot in 1890 was able to publish in the "Revue de Laryngologie" a well documented study based on 200 personal cases, many of which had excellent social results. All the same, antiseptic precautions at this time were not able to prevent some cases of severe infections which frightened surgeons into discarding this sort of surgery.

The second stage is that of fenestration of the posterior labyrinth. Otologists then had the idea of creating an artificial window to supple-

ment the deficient oval window. Little by little, we were able to create an opening on the posterior labyrinth disregarding the lesioned organs. Passow, in 1897, performed the first fenestration operation. Barany in 1910 set up a definite operative technique. Jenkins in 1913 used a burr to open the exterior canal and covered the fistula with a cutaneous graft. Holmgren in 1917 used the binocular microscope in his operative technique. Then Sourdille in 1928 with his three-staged technique obtained the first lasting results. In 1938 Lempert with his one-stage technique and using the endaural passage popularized fenestration.

This was then the birth of several new techniques which proved themselves, but which constituted attacking the posterior labyrinth which is only indirectly involved.

If however, after Sourdille and Lempert, the surgery of deafness corresponded exclusively to the simple "fenestration of the labyrinth," today discussions are opening on a much wider plane; it is the third stage in the history of this surgery which began with Rosen in 1952 and which is in fact what we have called "surgery of the windows."

Thanks to modern optical procedures which enable us to see the ossicles and windows perfectly magnified, and thanks to antibiotics which give an almost absolute guarantee against operational infection which was formerly so great, the otologist can reconsider the problem and try to come to a more functional and more delicate surgical conception instead of attacking the unaffected posterior labyrinth. The deficient organ is recuperated by directly attacking the affected organ, the stapes.

In this way, the authors seem to rediscover and apply former methods of mobilization of the stapes as a result of Rosen's publications (1952). But this surgery has already been surpassed, even in its conception and has been replaced by the more vast and veritable "window surgery." The action can be concentrated equally well on the stapes or on the otospongious lesion of the oval window when the neoformed bone blocks the stapedial footplate as on the round window when it is affected.

A certain amount of histological work has in effect been concentrated on the relative frequency of centers of otosclerosis around the round window. Guild, in 113 centers of otosclerosis studied in about 81 ears, notes that in 25 cases a focus affected the round window.

Nylen, out of 121 ears suffering from otosclerosis, points out that in 40 per cent of these cases such foci existed and that in 8 per cent they existed alone, the oval window being unaffected.

Nager and Meyer insist on the frequency of such foci second only in frequency to those far more classically localized around the oval window. Lindsay and Hemenway, in 37 otosclerosis ears, found 17 cases of foci localized at the round window, but concluded: "For the occlusion of the round window to lead to deafness it must be almost or totally closed." This fact agrees with the conclusions of Wever, who had already insisted on the difficulty of experimentally realizing an occlusion of the round window involving deafness.

Nevertheless, we do not possess any certain criteria, either before or during the operation, which confirm the localization of the otosclerosis foci, and only the statistical frequency at present guides the surgeon. We shall see later that our operative evidence indicates a relatively important frequency of round window lesions which could bring about an hypoacousia.

PATHOLOGICAL MODIFICATIONS OBSERVED DURING OPERATIONS

During the 80 operations being the object of this study, we found the following modifications in the windows.

MODIFICATIONS OF THE STAPES

The Branches. The stapes may appear normal in a relatively large number of cases (10 per cent). Lesions then figure exclusively on the annular ligament and on the circumference of the window.

In other cases it is the object of a simple thinning of the branches which, because of this, are less solid and make the surgical action directly on this bone more delicate though not impossible.

Sometimes the branches are so thin that they fracture spontaneously, either one or both, and generally near to the footplate of the stapes. The region of the fracture is occupied by fibrous ligaments constituting a pseudarthrosis which enables the head of the stapes and the branches to move easily, while the footplate remains fixed absolutely by the lesions.

In one case we found pseudarthrosis of the posterior branch against the footplate, associated with almost complete destruction of the interior branch replaced only by a long fibrous tract containing

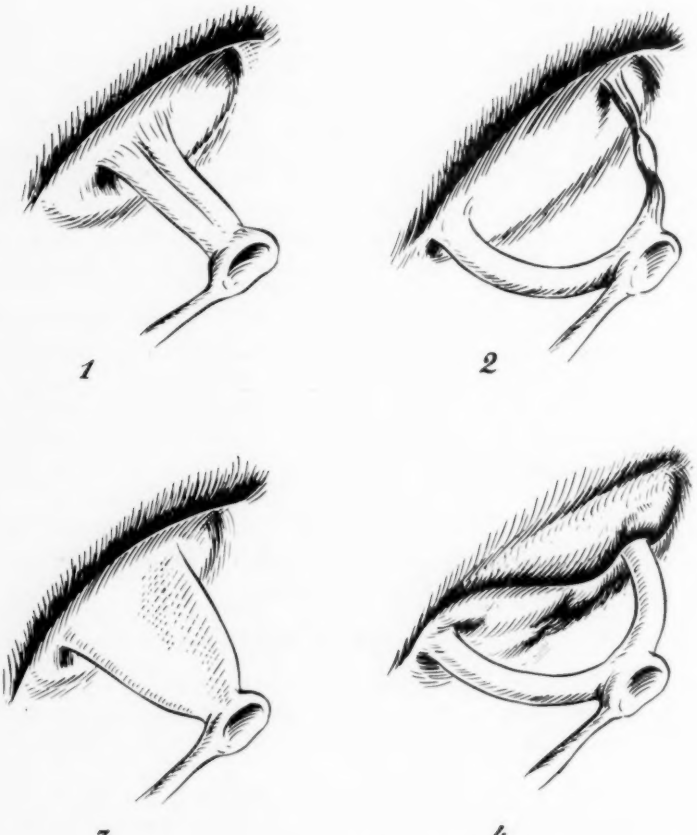

Fig. 1.—Some lesions of the stapes and oval windows: 1) Single branch joined to the immobilized footplate by a fibrous pseudarthrosis; 2) Disappearance of the anterior branch which becomes a simple fibrous ligament; immobilized footplate; 3) Stapes formed by a triangular block of bone; 4) Normal stapes' branches; considerable exostosis on the footplate of the stapes and on the anterior part of the oval window. The principal lesion originates in the relief of the facial nerve canal.

in its center a few ossified nuclei. In another case, we were able to note that the head of the stapes was joined to the footplate by a single median branch which was mobile as opposed to the footplate to which it was attached by a sort of pseudarthrosis. In a more precise microscopic examination, after removal of the fibromucous elements surrounding the branch, it appeared draped like the barrel of a shotgun. It is therefore logical to think that it represented the remains of two branches, interior and posterior; these must have been fractured spontaneously near the footplate, and due to osteoporosis they gradually approached each other on the median line and fused together in one single branch. This is, of course, an exceptional case, but it shows the importance in otosclerosis of the pathological troubles to which the branches of the stapes are subject.

In another case, the branches were completely joined by a thin flap of bone, the whole stapes forming a triangular bone, the top being the head of the stapes and the base joining the footplate.

The Footplate. The footplate of this bone is simultaneously fused to variable extents, which could join in multiple combinations with the different lesions of the branches. However, there appears to be a sort of balance of calcium, so that the footplate is found to be thick boned construction by exostosis, then the branches are thinner and more fragile. The footplate can be normal, the lesions being exclusively on the annular ligament. However, for more advanced syndrome, the footplate is rapidly modified. It is the object of exostosis and thickening which are usually localized in the anterior part but may from time to time affect the complete footplate as far as its posterior part.

Just as a balance exists between the exostosis of the footplate and the osteoporosis of the branches thinning of the median part of the footplate is frequently seen while its anterior portion is very much thickened. This thinning is shown by the blue transparent aspect which contrasts with the pear white of the footplate frame.

In two cases of resection of the footplate (where, as it was impossible to conserve the stapes, whose branches were spontaneously fractured and replaced by fibrous tracts), we were even able to estimate the pseudocartilaginous and soft consistency of this part of the footplate, which did not break when held by the forceps, but which afterwards bent back into its former position. These lesions are also connected to lesions surrounding the oval window.

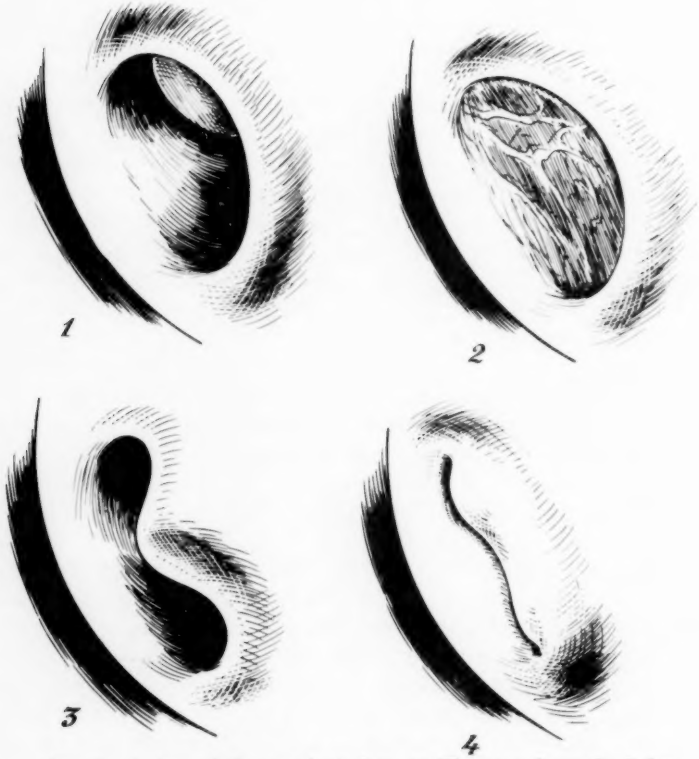

Fig. 2.—Lesions of the round window: 1) The normal round window niche; the membrane is seen at the back of the top; 2) The round window niche obstructed by fibrous tissue; 3) Round window niche partially obstructed by exostosis originating on the external edge; the rest of the niche was closed by fibrous tissue; 4) Total bone obstruction of the round window.

MODIFICATIONS OF THE OVAL WINDOW

The oval window is, in fact, itself the center of lesions corresponding essentially to the anterior part of the window, but more often the exostosis which one comes across makes its base the inferior base of the swelling of the fallopian canal immediately above where the anterior branch of the stapes arrives. The bone lesions enclose the anterior branch, reach the annular ligament and block the footplate.

It is often surprising to realize that very important lesions mechanically block the play of the stapes, enclosing its anterior branch,

but respecting the articular sulcus and the annular ligament. Sometimes they simply form one block with the affected footplate, the articular sulcus having completely disappeared.

MODIFICATIONS OF THE ROUND WINDOW

The round window is just as often the basis of large lesions. In 2.5 per cent of the cases a complete closure of this window by a bone tissue is seen, the niche of the window being now only represented by a light depression covered by an apparently normal mucus. It is only in scraping this mucus that a vertical crack is discovered—all that remains of the former bed of the round window.

Occasionally this bed is closed by a fibrous plug which takes up the whole space between the bone roof, the paries labyrinthica tympani and the secondary tympanum. In other cases the fibrous diaphragm totally closes the niche and the orientation of which is almost anteroposterior, while that of the secondary tympanum is frontal. This membrane may be partially open; it is dull, and fibrous tracts without any particular orientation are to be seen.

Finally, several processes might combine to close the round window—an exostosis beginning on the external border of the niche, transforming its normally oval opening into a form of hour-glass shape. The secondary tympanum, corresponding to the superior part of the hour-glass, may itself have its movement hampered by a fibrous formation closing the rest of the opening.

These microscopic lesions enable us to have some idea of how multiple the functional surgical act, with its diverse methods of restoration, can be.

THE TECHNIQUES OF FUNCTIONAL SURGERY OF THE LABYRINTHIC WINDOWS

All syndromes of otosclerosis which are connected with a veritable otosclerosis or with an ankylosis, which might be fibrous, may necessitate a surgical exploration of the windows.

However, it is apparent that false otosclerosis (i.e., blockage of the stapes by sequels of inflammatory lesions) does not give such favorable results as real otosclerosis taken at its beginning. Further, one must have on the functional plane a Rinne test which is sufficiently negative to give hope for an appreciable recuperation. In varied deaf-

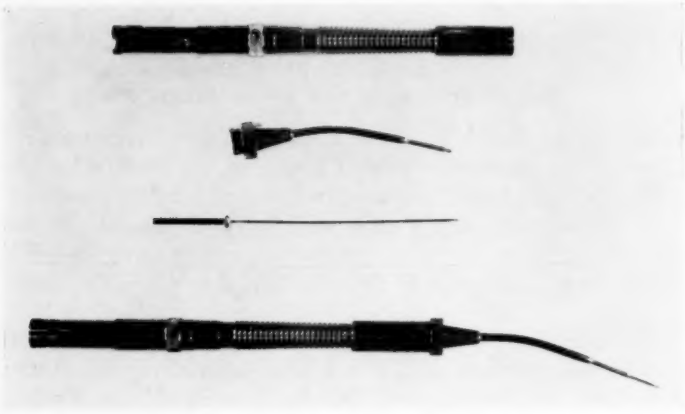

Fig. 3.—The microdrill especially designed for stapedovestibular osteotomy. At the top—the separate elements of the instrument: handpiece; rigid tube; very thin and flexible rotating element. At the bottom—the assembled drill. The extremity of the rotating element measures 2/10 mm and can turn at 10 thousand revolutions per minute.

nesses (i.e., when the anterior labyrinth is equally affected), a surgical action on the windows may be attempted if the Rinne test is sufficiently negative to warrant it; in these same cases corresponding to Shambaugh (Cases 3 and 4), it was preferable to avoid fenestration.

If the case is operable, only momentary contradictions might exist: extreme inflammation of the nose, the pharynx, of the eustachian tube and of the tympanum. On the other hand, all general infectious conditions, all general diseases constitute a susceptible element of momentary delay in the exploration of the labyrinthineal windows.

Since 1952 there have been numerous different techniques; several authors have described particular details concerning different instruments and different acts. Rosen, for example, has now evolved and described a clearly different technique to that exposed in his first works. We ourselves have, for several years, perfected his methods and have now arrived at a conception of a surgery with multiple technical aspects which would possibly adapt itself to each particular lesion connected to the round window, as well as to the oval window. The multiplicity of all these methods shows how full of evolution and transformation this surgery is.

THE PASSAGES

Three principal passages of access enable us to reach the region of the labyrinthical windows: the transtympanic, transmeatal, retro- or supra-auricular.

The Transtympanic Passage (Miot). We will simply cite this passage as being the one used by former authors. Miot mobilized the stapes through a tympanic incision parallel to the posterosuperior border of the bone frame, and therefore perpendicular to the radiant fibres of the tympanum which by way of their retraction enabled us to see the internal wall of the middle ear and the bed of the oval window. This passage is no longer used.

The Transmeatal Passage (Rosen). This is a passage frequently used nowadays. Special instrumentation has been devised in order to make the most of the binocular otoscope (giving a much more precise view than a magnifying glass) and to use the electric burr which is more rapid in the bone resection sometimes necessary especially along the posterior border of the tympanum frame which frequently conceals the mendostapedian region.

A local anesthetic is given through a posterior subperiostal meatus.

The incision is made right to the bone, one-half centimeter from the sulcus tympanicus (from one o'clock to six o'clock for the right ear), passing by the posterior wall of the duct. The flap is scraped from the exterior to the interior. The tympanum is detached at the level of the sulcus tympanicus from one to six o'clock. The tympano-meatal flap thus formed is pulled forward, being hinged by the axis of the malleus; the tympanicord, located and isolated, is also pulled forward. It can be cut if the patient suffers from buzzing.

Every anatomical disposition can exist making the access to the stapes more or less easy. Sometimes bone resection is then indispensable. Two or three quick strokes with a fine burr are given, sufficient to give the required access, holding in one hand the tympanum protector and in the other the hand piece. After functional movements, the flap is put back in its former place on the cruented surface.

The Retro- and Supra-Auricular Passage (M. Portmann). It no longer passes inside the meatus but above and behind the auricle.

Local anesthetic is by retro- and supra-auricular passage.

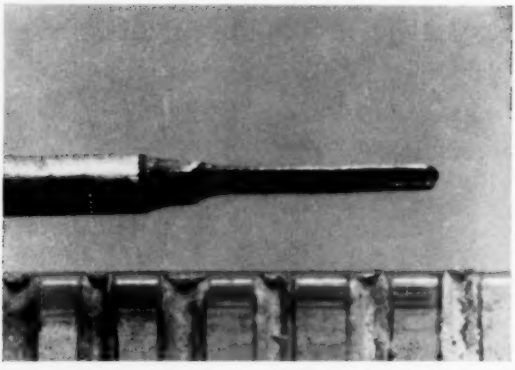

Fig. 4.—Extremity of the rotating element of the microdrill beside a millimetric scale.

The incision begins at the root of the helix, in front, passes between the tragus and the root of the helix, and then in front of the helix, joining the supra-auricular sulcus and finally the retro-auricular sulcus until the second third of this is reached. The meatus is thus reached and with a short bladed knife one cuts all around the meatus right to the bone.

Then the detaching of the skin of the auditive duct from the bone is carried out with separators, and microscrapers. Little by little the sulcus tympanicus is thus detached. Once this is completed the middle ear is reached. The resection of a portion of the external wall of the attic is always indispensable. It is carried out with a burr in a downward and backward direction so as to give a good view of the round window. The exploration of the functional surgical movements necessitated by the lesions are then carried out. If it was not necessary to make a tympanomeatal flap in the case of resection of the stapes' footplate the operative field is closed. A gentle drain by the external duct enables the replacing of the tympanomeatal sac. A closure with fine silk gut is used to close the incision.

In this way the transmeatic passage is very simple and gives rapid operative after-effects as the patient can leave within three days and sometimes even the same evening. But it gives less visibility and working possibilities on the stapes and oval window.

On the other hand the retro- and supra-auricular passage gives a wider access to the lesions but necessitates a little drain, a dressing and longer hospitalization. This is the passage we prefer at the moment. It gives better conditions for working on the stapes and windows. In case of failure to mobilize the stapes and if the round window is normal, it allows the opening of the oval window by resection of the affected footplate, or the possibility of immediately completing the operation by a classical fenestration.

The transmeatic passage is, all the same, very often chosen either for psychological reasons (the patient unwilling to support any other incision) or for technical reasons (e.g., when the second ear is operated on). In fact, the lesions are known to be symmetrical, if the operation on the first ear was very easy, the other ear will be operated by transmeatic passage which is, of course, much easier.

EXPLORATION AND ACTION ON THE LESIONS

These include surgery of the round window, surgery of the stapes and oval window.

a) *Surgery of the Round Window.* The exploration is carried out, directing the microscope downward successively to the promontory, and beneath and behind the niche of the round window are seen.

If this window is normal the secondary tympanum appears in a frontal plane in relation to the patient (i.e., very obliquely and almost in the same plane as the observer's optic ray). It is pale, bluish and transparent. If, on the contrary, the niche of the round window is subject to some pathological process, as those described above, a surgical act is then necessitated. Using magnifying power 16 or 25, it would be advisable to remove the diaphragm and fibrous blocks with little curettes and little spatulas especially adapted to this work.

b) *Surgery of the Oval Window and the Stapes.* This operation can be directly on the oval window itself or indirectly on the head of the stapes. Different authors have tackled this problem (S. Rosen, Goodhill, Myerson, Fowler, etc.).

These are the processes we utilize, having acquired a wide passage of access by necessary bone resection and the exploration of the lesions, it is immediately seen by the importance of the latter, if the footplate can be mobilized by means of the head of this bone, or if, because of the considerable exostosis at the level of the oval window

and of atrophy of the branches, a more direct act is required on the lesions.

Simple Mobilization. All the types we have come across have been described under pathology. If the case seems simple, mobilization can be tried at the level of the neck of the bone with a mobilizer according to the technique described by Rosen. In the case of a failure, one should not insist, as one would risk impairing the finer action of the other operations in fracturing the branches.

Another method which we readily utilize consists of dislocating the incus and the lenticular bone from the head of the stapes without completely disjointing it, to puncture the center of the stapes head with a very fine point and then to give this latter anteroposterior movements by pressing gently towards the oval window. If this fails, one should dislocate the incus completely from the stapes giving a much wider view of the footplate and on the oval window.

Then one works directly on the footplate, using pressure on the rim near the annular ligament at the top and bottom forward and backwards, these maneuvers being alternated before the mobilization of the stapes if the lesions are not very important. Suddenly the stapes is seen to move; it is then very important to estimate the solidarity of the complete bone, to verify whether the footplate moves when pressure is applied to the head and the opposite. In the opposite case, one must immediately verify a probable fracture of the branches.

Pressing on the head of the now mobile stapes one can see whether the two windows have normal play by watching simultaneously the secondary tympanum and the round window follow this movement in the opposite direction. It is a fundamental test, for it enables one to be certain whether the stapes functions or not.

Stapediovestibular Osteotomy. Should the preceding maneuvers give no results, one must refrain from mobilization by the described means before the fracture of the branches, and go directly on to the lesions, the veritable stapediovestibular osteotomy.

To facilitate these direct maneuvers, a special instrument has been devised—a microdrill, the extremity of which is 0.2 mm thick and can turn from 8 to 10,000 revolutions per minute. With the help of this instrument the bone lesions are directly tackled (those which more often than not join the anterior part of the footplate to the frame of the window).

Then the stapedian mobilization should present no difficulties. It is wiser to revert again to the burr to finish off an inadequate bone resection than to insist on a premature mobilization. If not, one runs the risk of fracturing the stapes branches, thus impairing the functional result. In the case of a total ossification of the annular ligament, one must somehow cut a new footplate with the burr, perforating all round the edge of the footplate. The play of the windows is thus verified as mentioned above.

Reopening of the Window and Resection of the Footplate. In some cases the stapes is physiologically irrecuperable, either because the branches have disappeared or because the extent of the bone lesions around the window and the footplate impair the result of an eventual osteotomy. It is therefore possible, if the round window appears to be unaffected, to remove the footplate from the injured stapes so as to effect a true fenestration of the vestibule at the most suitable place, the region of the oval window. This is not a new idea as authors of the nineteenth century who practiced the mobilization of the stapes, even at that time executed its ablation and in certain cases that of the footplate too. We propose the following process:

Arriving at the ossicles and the windows, the round window is freed to the desired extent. Mobilization of the stapes is done according to the different processes. In the case of impossibility or failure and if the round window is not obtured by a block of bone, in the same operative stage, it is decided to carry out the resection of the affected stapes and its footplate (i.e., the opening of the anterior labyrinth).

So that the tympanomeatal flap is sufficiently freed and supple, and so as to be able to bring it to the level of the circumference of the window which is deeply set between the reliefs of the first cochlear spiral turn at the bottom, the region of the window is cleansed, removing the mucus all around, and especially on the Fallopian canal, the back of the first spiral and at the back and front of the window.

If the relief of the pyramid is too prominent, it is possibly rectified by a few touches of the microscopic burr. Then the other ossicles are resected; the incus which is easily passed to the operative field without opening the attic; the malleus which is completely resected to give the tympanum a great suppleness; it is very easy with patience to separate the handle of the malleus from the tympanic membrane without perforating it.

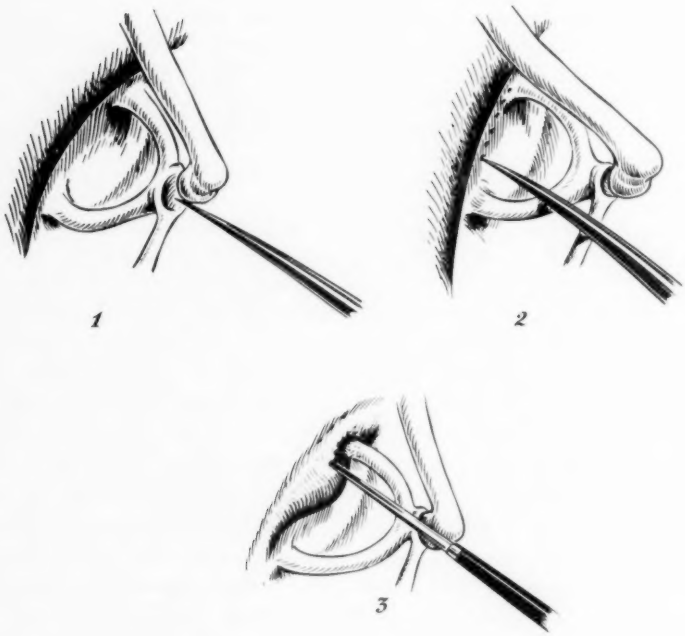

Fig. 5.—Some stapedial maneuvers: 1) subluxation of the incus: mobilization of the stapes by pricking the head with a point; 2) stapedovestibular osteotomy at the point; 3) stapedovestibular osteotomy across a bone lesion with the microdrill.

Then the tympanomeatal flap is prepared. Two vertical incisions are made in its exterior edge, the one to the inferior posterior part of the meatus, and the other on the anterior part, forwards from the level of the anterior spina and progressing obliquely downwards towards the middle of the tympanal frame, and culminating clearly forwards in relation to the Schrapnell. The prepared flap is lain forwards and downwards while waiting to be used.

The resection of the stapes and the opening of the oval window is effected with a microdrill when the lesions are large, so as to separate what corresponds to the former footplate from what corresponds to the frame of the window.

With extremely fine hooks which are passed into the vestibule under the piece of bone which is to be resected with a strong magnification (25) and with much patience one is able to free the footplate.

So as to close the oval window which has been opened in this way, the tympano-meatal flap is pushed in its direction. This flap will close; the tympanum at the bottom by lying against the upper edge of the first turn of the spiral, the atticus above by fixing itself to the Fallopian canal, then on what remains of the external wall of the middle ear.

A small plug soaked in antibiotics is placed across the flap against the window so that the conjunctive surface is applied to the circumference of the latter, then a meche is applied. The external cutaneous incision is sutured with fine silk gut.

This operation, of course, gives way to more important after-effects than the simple mobilization of the stapes. In the days following the operation, the patient suffers from a serous labyrinthitis which will cause a momentary loss of primary auditive improvement, together with a sensation of instability which will disappear after four or five days.

RESULTS AND IMPORTANCE OF THIS SURGERY

Of the 80 patients submitted to this operation we obtained 60 per cent appreciable gains, reaching as far as 10 to 55 decibels, which were clearly perceptible to the patient. Sometimes we obtained complete restoration of the Rinne test and an absolutely normal level of hearing.

When one analyzes the different statistics it is interesting to note that as the authors utilize improved techniques, the percentage of good results gradually increases. This fact is especially apparent when the same surgeon is responsible. Thus, Rosen notes 37.3 per cent successes for his first technique (direct action on the neck of the stapes), and 50 per cent of good results for his second technique (direct action on the footplate and fenestration of the footplate). It seems, therefore, that as the technical possibilities of these operations improve the successful results will increase. However, the far-reaching results are not always lasting.

In four of our cases, the result obtained has failed in a few months. Temporary improvement could always be obtained by operating again.

In one other case, the gain has partially decreased in three months at a level which is still socially acceptable and at which hearing remains.

The opposite phenomenon happened in two patients who obtained no improvement but during the following three months felt their hearing improve, then stabilize after a gain of 30 decibels.

It appears that bleeding is especially harmful for ultimate results. On the other hand, the conjunctivo-vascular process which is the cause of otosclerosis presents an evolutive character variable according to patients. In some cases, it appears that mobilization of the stapes increases the lesions. One of our four failures mentioned above presented after the first operation a spectacular gain which decreased in 10 months; a second spectacular gain after the second operation decreased in three months; finally, the third operation gave a completely joined stapes which could not be mobilized.

To fight against this evolution we systematically tried several processes:

The placing of a little plug of resorbable substance soaked in cortisone against the mobilized stapes.

Tubal insufflations under pressure during the weeks and months following the operation.

Pneumatic massage of the tympanum by the duct passage.

Ultrasonotherapy by the transmeatic passage (L. J. Barbe).

Cortical hormonotherapy was also recommended for some patients, 10 to 20 milligrams of cortancyl daily for several weeks and months.

If an allergic case seems probable (association with eczema, asthma, hay-fever, etc.), a specific desensitization was undertaken before and after the operation on the ear.

Unfortunately, up until the present, it seems that when there is an evolutive process, no maneuvers, no therapeutics can stop the evolution of the disease in a definite manner.

Surgery of the windows should not be opposed to fenestration as too many authors seem to think. The one operation completes the other. But the mobilization of the stapes should always be carried

out before fenestration because the latter is a more important attack and carries risks, whether it is a classical fenestration or the opening of the oval window.

This surgery of the windows, thanks to antibiotic protection, the use of the microscope and increasingly finer instruments, will, in the ensuing years, become even more perfected. It is already permissible to think that it will supplant the classical fenestration of the semicircular canal in a more or less permanent way.

45 Cours de Maréchal Foche

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IV

MECHANISMS OF DISABILITY OF SPEECH RESULTING FROM POLIOMYELITIS

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The impairments of speech which are familiar in acute bulbar poliomyelitis commonly persist.[1-5,6] There is comparatively little report of this aspect of poliomyelitic disability,[7,8] though the abnormality of speech may be equal in severity to that of other pathological processes (such as anomalous cleft of the palate) which have received much scientific attention and for which speech rehabilitation measures are presumed to be essential.

Clinical experience is here reported with a group of severely involved bulbar and bulbar-spinal poliomyelitis patients. A classification of these disabilities of speech has been derived upon the basis of clinical and roentgenographic observations. In patients of this group the mechanisms of disability were commonly multiple and mutually exaggerating in their effect.

There has been found a surprising variety of problems related to speech performance. Conversely, several compensatory mechanisms have been detected, and others therapeutically encouraged.

Speech therapy has been found to be integrally related to measures designed to restore performances of ingestion of food and control of respiration, and the general role of speech therapy in rehabilitation of paralytic disability in this region is discussed.

The subjects of this study were a group of 47 patients, who were under observation in a general program of evaluation and trial therapy

Aided by a grant from the National Foundation for Infantile Paralysis.

of residual disability resulting from bulbar poliomyelitis.[1,3-5] These patients demonstrated relatively severe bulbar poliomyelitic disability.

In addition to previously described procedures of clinical and roentgenographic observations of motor performance in the bulbar areas,[3-5] the patients' speech was observed by clinical methods standard in the field of speech pathology and, in selected cases, by auditory and visual analysis of recorded speech performances.[9]

The clientele has been described in other reports.[3-5]

CLASSIFICATION OF MECHANISMS OF DISABILITY OF SPEECH

Deficiency of Respiration. During acute bulbar poliomyelitis, there is commonly a loss of speech due to a complex of causes, including respiratory deficiency, the continuously open tracheotomy, filling of the larynx and pharynx with accumulated secretion, and the systemic prostration and possible encephalitic components of the disease.

In early convalescence, the patient may not be expected to speak, and this situation may specifically be explained to him in terms of the tracheotomy, or of some other mechanism. Principal efforts of motor rehabilitation are commonly directed to parts other than the bulbar area. Many patients adapt to this circumstance by accepting muteness and depending upon visual or manual signals for communication. Others devise characteristic nonvocal lingual and labial sounds. Initial efforts of vocalization may produce coarse, guttural sounds which are further obscured by secretions accumulated in the pharynx and larynx; an appropriate reaction of the patient is to not employ these foreign sounds for verbal communication.

With resumption of speech during convalescence from respiratory ventilation deficiency, the patient's awareness of this deficiency may be re-emphasized as he finds that he is unable to accomplish the increased respiratory work required for the intermittent valving of the respiratory stream which constitutes the basis of phonation. In this circumstance, they may become habituated to a voice of minimal intensity, supplemented by grimaces and gestures. These patients may become markedly dependent upon a patent tracheostoma and, for this reason also, accept a deficiency of vocal intensity in favor of a completely or partially open tracheostoma. In this latter circumstance speech encouragement and specialized therapy are particularly effective in adapting the patient to the pharyngeal portal of respiration. An interesting observation, made particularly in one of the subjects who

had had an open tracheotomy for two years, was that of a loss of the normal interspersing of inspiration with speech. She would characteristically inspire deeply through the open tracheotomy, place finger over the tube, converse to the limit of the succeeding expiration, and repeat inspiration through the tracheostoma. The speech pathologist was able to retrain the patient to intermittent partial inspirations via pharynx and larynx appropriately interspersed in conversational speech. The technique was apparently of particular effectiveness in relieving this patient of dependence upon the tracheostoma.

An additional circumstance of complete aphonia was that of persistent occlusion of the pharynx by apposition of its anterior and posterior walls between the lordotic cervical spine and the retrodisplaced tongue, larynx, hyoid, and structures of the inferior portion of the face, as illustrated in Figure 1*a*.[5]

Abnormal Function of the Larynx. During the acute bulbar poliomyelitic illness, the pharynx and larynx are commonly filled with secretions. With initial recovery of speech, the extent of its disability becomes more evident. When the pharynx and larynx airway is again patent and is penetrated by a respiratory stream, so that speech can be evaluated, it is commonly found to be lower in pitch and to be of diminished range. The tone is typically irregular with a tremulous quality of attempted sustained sounds.[9] An intriguing preliminary clinical observation in three patients having disseminated weakness in the facial musculature as well as in the larynx was that of a synchrony of the intermittent bursts of sound in sustained tone with tremor in the area of the weakened orbicularis oris or orbicularis oculi muscles. This grossly observed synchrony has not been further evaluated by objective recording methods.

The respiratory stream may be poorly controlled by the larynx for purposes of speech, with audibly apparent excessive penetration between the insufficiently adducted vocal cords, giving a "breathy" tone. A common finding was that of attempted louder voice, as indicated by increase in effort of respiration and appropriate motions of the face, the voice was actually diminished in volume. It was noted that this particular manifestation of disability was commonly associated with a deficiency of cough.

Two peculiar forms of abnormal co-ordination of the larynx were also observed. In one adult woman seen 17 months following severe bulbar poliomyelitis, an obstruction to effortful inspiration was noted.

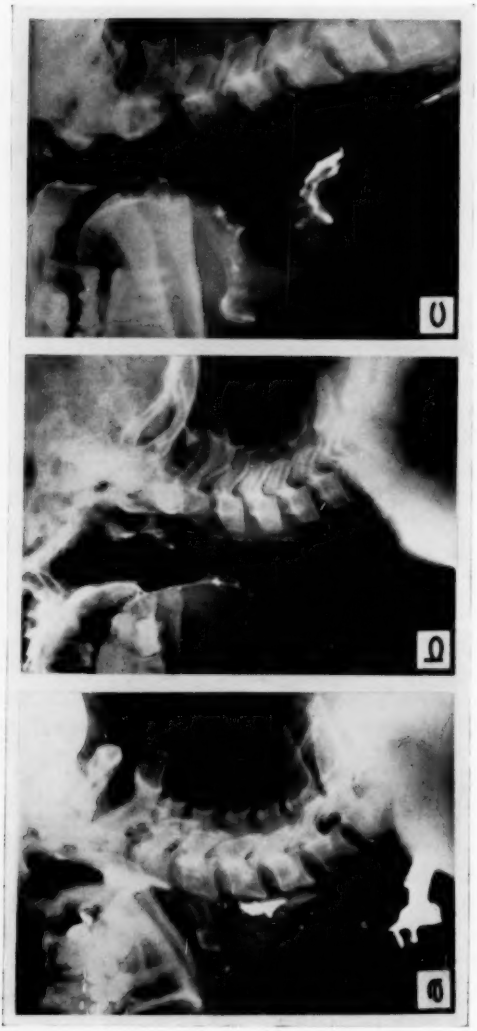
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FIGURE 1

a. Occlusion of the pharynx. Lateral roentgenograms of pharyngeal area of a woman, 15 months following acute poliomyelitis at 39 years of age. There is residual severe weakness in the bulbar and upper cervical area. The pharynx is occluded by apposition of the tongue, hyoid, and larynx to the lordotic cervical spine. The lateral processes of the hyoid bone extend on either side of the body of the fourth cervical vertebra.

b. Subject 54 months following acute poliomyelitis at the age of nine years. There is residual severe weakness of the entire constrictor and suspensory musculature. Nasal respiration, mouth closed. Note expansion of the palatopharyngeal isthmus, the meso- and hypopharynx, with inferiorward and anteriorward displacement of the tongue, hyoid, and larynx.

c. Expansion of pharynx incident to effortful phonation. Adult patient, 24 months following acute poliomyelitis. There is marked anteriorward displacement of the hyoid and larynx, with dilatation of the hypopharynx and vestibule of the larynx and lesser dilatation of tracheal and cervical esophagus.

On laryngoscopy, the right vocal cord was found to be in partial adduction. During quiet inspiration, the vocal cords approximated slightly and during increasingly effortful inspiration this approximation was increased to point of near apposition in the midline. Appropriately, the patient became increasingly anxious with greater efforts of respiration and, when obstruction was severe, it was necessary to remove the covering of the tracheotomy tube. This direction of vocal cord motion during inspiration is the converse of that observed laryngoscopically in normal subjects.[10,11] Freystadtl has observed similar paradoxical motions, which he termed a "functional inspiratory glottic spasm."[12]

In one patient there was an abrupt occlusion of the larynx during effortful speech, interrupting expiration. The larynx was persistently totally occluded despite strong respiratory efforts for periods of from a few seconds to one minute or longer, necessitating resumption of respiration via tracheostoma. The onset of this adduction was accompanied by an audible "click" sound in the voice. With prolonged intensive speech retraining, this peculiar occlusion diminished progressively, though for months afterward an occasional abrupt, quick, and brief pause was still detectable in voice, and in phonatory effect this was apparently identical with the onset of the earlier noted abrupt, severe, prolonged occlusions. During the weeks of severe manifestation of this occlusion, the patient had learned to accomplish relief by a swallow effort. Unfortunately, it was not possible to accomplish indirect or direct laryngoscopy in this patient during the period of our observation by reason of severe residual contractures in the area of mouth and pharynx.

These episodes apparently are identical with laryngeal crises reported in tabes,[13] and with the tonic phase of seizures induced by cerebral cortical stimulation in the monkey.[14] They differ in abruptness from the "functional phonatory glottic spasm" described by Freystadtl.[12]

One patient was observed to have bilateral paralysis of abductor musculature of the larynx, of such degree that the cords were near apposition at the midline. To our knowledge, this is the first patient in whom this pattern and degree of disability were found following poliomyelitis, though a similar effect was seen by Roy[15] following an influenza-like illness.

Observation of motor function of the larynx by direct or indirect laryngoscopy is not always possible in early convalescence by reason

of residual secretions in larynx and hypopharynx, or of contractures limiting mobility of the jaw and of the column arrangement of tongue, hyoid, and larynx. In such circumstance, roentgen planography of the larynx at rest or during sustained tones[16] has been helpful in evaluation of relative position of the vocal cords.

Impairment of Suspension of the Tongue, Hyoid and Larynx. As an effect of contracture of the muscles suspending the tongue, hyoid, and larynx, the pharynx and oral cavities may be compressed by displacement of these structures upward and dorsally,[5,17] as shown in Figure 1*a*. As an effect of weakness of these muscles, and possible contracture of the sternohyoideus and sternothyroideus in subjects prolongedly tracheotomized, the pharynx may be expanded by displacement of the column ventrally, or the pharynx and oral cavity may be expanded by displacement of this column ventrally and downward (Fig. 1*b*). Thus, all or a part of the "resonant tube," as defined by Kelemen,[18] may be extensively modified in volume and contour.

During speech performance, this expansion of pharynx and oral cavities may be increased, probably further distorting the resonance qualities (Fig. 1*c*). Again, this may result from further anteriorward or anteriorward and inferiorward displacement of the tongue-hyoid-larynx. The extent to which this displacement is passive, by increase of pressure of the contained air, and to which this results from disproportionate motor action of the muscles suspending the tongue, hyoid, and larynx from the mandible, compared with suspension from the base of the cranium, is not distinguishable by external visual or roentgen observations of these motions. Evaluation of strength in these muscles requires observation in other maneuvers, such as in swallowing.[4,5]

During speech performance, there also may be compensations for this expansion by extension of head at neck, thus accomplishing elongation of the pharynx and diminution of its transverse diameters, or tilting or tipping of the head. Apparently, these speech-associated maneuvers begin early in convalescence at time of effortful phonation, and may be retained when no longer required by the former circumstance of disability.

Gross displacement of the tongue-hyoid-larynx column is commonly associated with weakness of the constrictor muscles, thus allowing further expansion of the pharynx by lateralward dilatation. This evaluation is dependent upon roentgenograms in sagittal plane.

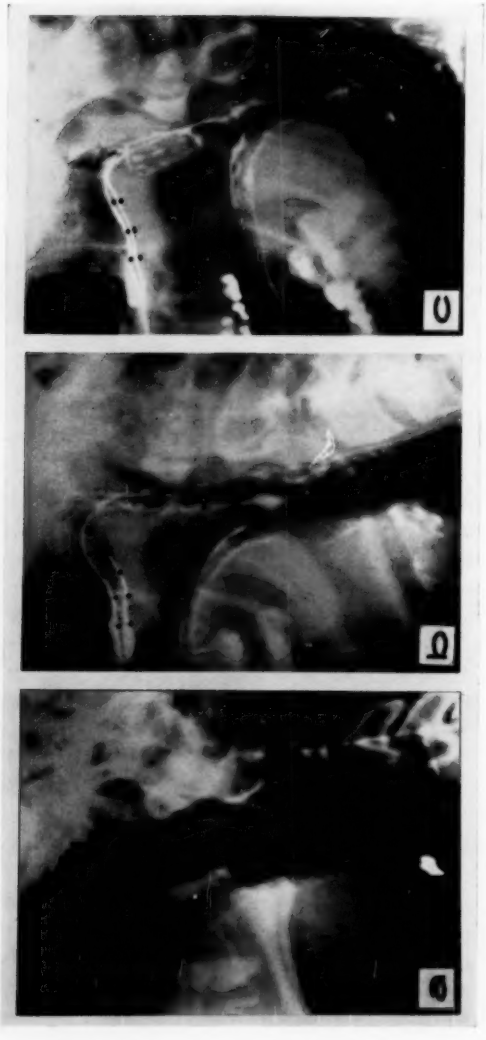

FIGURE 2

a. An adult male, 24 months following acute poliomyelitis. There is marked weakness of the levator veli palatini and suspensory muscles of the tongue-hyoid-larynx column. The palate is lax and pendent. The tongue and hyoid (at square) are displaced downward and forward.

b. Adequate performance of palate in circumstance of residual weakness of the levator muscles. The subject is an adult woman, 39 months following acute bulbar poliomyelitis and subjected to intensive speech retraining efforts for approximately one year following her acute illness. Lateral roentgenogram was obtained during phonation of moderate intensity in the medium range of pitch. The palate is indented upward in its midportion and drawn backward to firm approximation to the posterior pharyngeal wall. The lines of matching dots indicate separate lines of radiopaque medium outlining the upper level of the palate on either side. The difference in level of these lines gives approximation of difference in the degree of elevation of the levator muscles.

c. The same subject as *b*. There is phonation of moderate intensity in high pitch. The palate is drawn sharply posteriorward with its upper surface nearly parallel with that of the hard palate and its posterior face in close apposition to the posterior pharyngeal wall. It is noted that the two lines of radiopaque medium on the upper surface of the palate, again indicated by paired dots, are now in closer approximation.

Additional mechanisms of compensation are observed in circumstance of weakness of the pharynx constrictor muscle. Thus, with asymmetrical weakness of the pharynx constrictor, the posterior wall of the pharynx and a portion of the lateral wall on the side of the greater weakness may be seen to be drawn transversely toward the stronger side—the "curtain movement" of Vernet.[19] The otherwise lax or distended side of the pharynx is thus diminished in area. Though these various displacements of the pharynx and oral cavity at rest may be defined by standard roentgenograms, their further distortion during speech requires fluoroscopy or cinefluorography.

In common association with the paralytic expansion of the hypopharynx, there was a pool of secretions and traces of bolus retained from recent swallows. This was manifested by a "bubbling" quality of the speech, which was at times conspicuous to the observer, though apparently unnoticed by the subject. The bubbling sounds were diminished momentarily by interposed swallowing and could be substantially diminished during test periods in therapeutic conversation by repeated swallowing. There was, however, little apparent profit in attempts to make incidental swallowing more frequent, and the speech therapist was at the risk of merely heckling the patient. This symptom was substantially increased during acute respiratory infections.

Abnormal Patency of the Palatopharyngeal Isthmus. The most common disability of speech in these patients was that of abnormal patency at the palatopharyngeal isthmus, resulting in nasal distortion of speech. This nasal distortion of speech was associated most commonly with a visible deficiency or asymmetry of elevation of the palate. The corresponding lateral roentgenograms are shown in Figure 2. In other patients, this nasal quality of voice was found in association with a deficiency of action of the superior constrictor muscle converging the posterior and lateral pharyngeal walls at the upper rim of the isthmus, as evaluated roentgenographically. In the circumstance of gross deficiency of the palate levator muscles, so that the palate could not be carried upward to its normal range in speech,[20-22] or of weakness of the superior constrictor, there was a gross nasal distortion of all components of speech. This closure of the upper rim of the isthmus in phonation is well distinguished in poliomyelitic impaired as well as in normal patients from the median approximation of the palatopharyngeal folds, as found in inflation of the combined oral and pharyngeal cavities.[3] In no instance was this latter manner of inflation found as a compensation for deficiency of the speech-appropriate closure of the upper isthmus rim. Correspond-

ingly, the nasality of voice was inconstantly combined with nasal regurgitation of food in transit through the pharynx, for the mechanism of closure of the palatopharyngeal isthmus is the same in oropharyngeal inflation as in swallowing.[3]

In some of the patients having severe and disseminated bulbar impairment and whose pharynges had been disused incident to gavage feeding and tracheotomy during their acute illness, there were contractures of the musculature of the palate. These could be distinguished by clinical maneuvers, such as digital pressure upward and posteriorward on the midpalate, in the direction of elongation of the tensor veli palatini, or upward and forward upon the inferior edge of the palate, in the direction of elongation of the palatopharyngeal muscles. In either circumstance the presence of contracture would be indicated by palpation impression of disproportionate resistance or by the subject's report of pain. In two patients a prominent symmetrical transverse ridge was seen to protrude from the superior-anterior aspect of the palate. The direction of this ridge was that of the tensor veli palatini muscles within the soft palate. On palpation, it was of less passive mobility than the adjacent portions of the soft palate, and upward and posteriorward pressure elicited moderate pain within the general area of the palate.

Deficiency of Lingual Articulation. Deficiency of lingual contribution to speech may result from inferiorward displacement of the tongue or from impairment of the muscles of the tongue itself. The general displacement may result from deficiency of musculature supporting the tongue and hyoid from the base of the cranium and the mandible, in which circumstance the posterior portion of the tongue is lacking in approximation to the soft palate and the posterior portion of the hard palate. This was found in association with general enlargement of the pharynx, by the same mechanism. In the circumstance of marked weakness of the musculature suspending the mandible, the entire tongue is displaced inferiorward and, again, the articulation of speech by intermittent approximation of tongue to the upper teeth and osseous palate cannot be accomplished.

Poliomyelitic impairment of the lingual musculature is of dual pathologic effect, for the muscle so involved becomes conspicuously atrophic and the mechanical effectiveness of the lingual mass is diminished in this manner, as well as by the loss of effectiveness as the result of paralysis per se (Fig. 3).

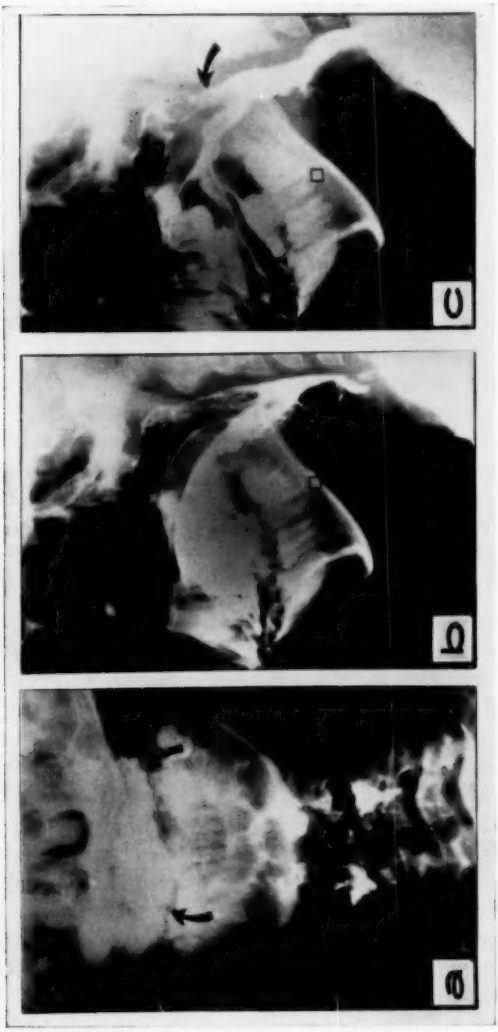
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FIGURE 3. Abnormalities of Swallowing in Poliomyelitic Impaired Subjects

a. Posterior-anterior view of bolus in the preparatory position. Subject, adult male, seven months after acute poliomyelitis. Asymmetry of the inferior border of the bolus, reflecting weakness and diminution of mass of tongue on left side, in film.

b. Lateral view of bolus decanted from mouth into pharynx. A marked and irregular diminution in the mass of tongue is shown by the irregular contour (at series of dots) of the inferior border of bolus in the mouth. Weakness and diminution of the mass of tongue is shown further by irregular lingual border of a portion of bolus in the mesopharynx.

c. Later stage of swallowing in subject *b*. Note persistence of the retained mass of the medium in a pocket of arophy of the tongue. The hyoid (at square) is now moved cephalad and ventrad, the posterior wall of the meso- and hypopharynx is moved ventrad, and the hypopharyngeal sphincter is widely open. At this stage of swallowing, the palate is normally elevated, but in this patient remains lax and pendent, reflecting weakness of the levator veli palatini. Incident to constriction of the pharynx at this stage, there is regurgitation of the bolus upward in the palatopharyngeal isthmus (at arrow), despite the open hypopharyngeal sphincter. This indicates weakness of the constrictor at this level.

Deficiency of Articulation at Lips. Labial, like lingual, articulation depends upon appropriate relations of the skeleton of the face, as well as adequacy of the labial musculature. In the circumstance of abnormal dependent position of the mandible, labial occlusion cannot be accomplished. Actually, the severe involvement of the muscles of mastication, allowing displacement of the mandible, was commonly associated with weakness of the labial muscles, and constituted a circumstance of exaggeration of their disability.

Paralytic impairment of the labial muscles occurs in a striking variety of patterns, selectively affecting the portion of the orbicularis oris which is in encircling relation to the lips, the muscles originating in the lateral portion of the orbicularis, or generally affecting all of these.[9] Weakness of the orbicularis and of the radial muscles was mutually exaggerating in its effect. Combined weakness of the ipsilateral orbicular and radial muscles resulted in laxity and dependence of the lateral margin of the mouth at rest, with external spill of saliva and traction of the impaired toward the more normal side during activation, possibly with paradoxical opening on the stronger side. In patients having gross persisting patency of the lips, labial effort during speech and ingestion might be given up, the lips becoming unmoving in disproportion to their actual capacity for motion.

COMMENT

The principal clinical problems of the patient having acute bulbar poliomyelitis or disability persisting after this disease have been those of survival and recovery of performance of the pharynx in deglutition and of the pharynx and larynx in respiration. The disability of speech commonly associated with the acute phase of this disease has been utilized as criteria of its diagnosis, but the persisting difficulties of formation and modulation of speech are commonly considered ancillary to the principal clinical situation.

The clientele of this study had generally had severe and disseminated poliomyelitic disability of the bulbar area. This disability was compounded of multiple factors, including not only denervation by destruction of motoneurons governing the suspensory and intrinsic musculature of the bulbar viscera and the muscular atrophy and contractures appropriately associated with this denervation, but also co-ordinative disabilities of the larynx, which are of obscure origin central to the motoneurons. In the latter category are the distortions of larynx motion during inspiration and during expiration noted in this report, and of the tongue as described by Canestrini.[23] These

general co-ordinative disabilities may well be related to the focal areas of neuronal pathology found by Baker, Matzke and Brown to be disseminated through the medulla.[24]

There is much interdependence of the various motor elements in the bulbar area. The role of the suspensory muscles of the palate and of the tongue, hyoid, and larynx in placing these structures in position of best function has been emphasized in this clinical description. The importance of these supporting muscles to speech performance of the larynx and pharynx has been emphasized by Kenyon[25,26] and by Griesman.[27] The positional support given the larynx by the musculature of the hypopharynx is mentioned by Pressman.[11] A significant factor of exaggeration of disability in these patients having disability of the lips, tongue or palate resulting from their displacement was that of their habitual disuse of these articulation factors of speech. Subsequent rehabilitation of their motor contribution to speech requires their more normal reposition, by the use of the supine position, or by prosthetic or other mechanical support.

With recognition of these mutually exaggerating mechanisms of impairment, it has become increasingly apparent in this project that the optimal time of initiation of speech rehabilitation efforts is in the immediate postacute period. These efforts should be initiated even though speech performance is markedly limited by reason of the greater respiratory effort required as the result of accumulation of secretion in the pharynx and larynx, and of the common circumstance of inadequate adduction of the vocal cords and the resultant spilling or wastage of the expiratory stream. The early introduction of speech therapy will result in utilization of the larynx, pharynx, tongue, and lips at a time of neuromotor recovery when function is highly contributory to rehabilitation. This early resumption of speech justifies particular effort on the part of the rehabilitation team to restore respiration to the pharyngeal route, even though this be only in expiration, as by the use of the valved tracheotomy tube.

Early institution of speech therapy should be prophylactic of development of alternative modes of communication, as by sounds generated within the mouth or by grimaces, gestures, and hand signals. Verbal communication will thus continue to be familiar and habitual, and the patient may be expected to have less concern and anxiety about distortions of the newly recovered speech, as at these times these distortions can more readily be attributed to incomplete recovery.

It is essential that the speech therapist and other members of the rehabilitation team undertake the task of speech rehabilitation in severely disabled poliomyelitic patients with sufficient respect for the duration of effort indicated. Also, it is essential that periodic recording of speech be made for purposes of evaluation. This rehabilitation effort should also compose the basis for decision of the surgical correction of residual impairments, such as the King or the Kelly-Woodman procedures for achievement of persistent abduction position of the abnormally adducted vocal cord.

The natural temptation is to discontinue these considerable efforts when seemingly adequate speech is achieved. Later, however, the patient may regret residual disabilities of this performance when he eventually comes to measure his abilities in comparison with those of normal associates rather than the convalescent clientele with whom he had previously compared himself. It may be necessary for the speech clinician to emphasize to the patient the considerable importance of the best recoverable speech in his postconvalescent daily living, as he returns to his family, social, scholastic, and occupational environment. The continuing objective of the rehabilitation personnel, and of the speech therapist in particular, should be that of attainment of speech as near normal quality as possible. The extent and duration of effort, and competition of concern of the patient with disabilities other than speech may tempt the patient and his family and professional associates to accept a partial, but unnecessary, disability of speech. Therapeutic termination or attenuation is best avoided by rehabilitation in speech which is well-balanced with parallel activities concerned with recovery of respiration and gross motor performance.

In our rehabilitation efforts, speech therapy was intended to be more than the facilitation of communication. In spontaneous or guided speech efforts, the patient is utilizing motor system of the entire bulbar area. This rationale of intensive speech therapy, which is familiar to those engaged in motor rehabilitation in other parts of the body, is that of diffuse or synkinetic participation of the musculature of the region in each of its varied motor functions. This diffusion of participation has been found by Doty and Bosma[28] to be particularly conspicuous incident to such bulbar performances as swallowing and participation in effortful respiration in the monkey. A general perspective of aid to the speech therapist is that of the secondary derivation of laryngeal phonation from the more primitive laryngeal valving of respiration[11] and of speech articulation from elements of ingestion.[29]

Appropriately the speech therapist should, therefore, share the general objective of the rehabilitation personnel concerned with function of the mouth, pharynx, and larynx, in general, aimed at achievement of its normal mobilization and effective regulation of ingestion and of respiration, as well as of speech. As a strategic member of the rehabilitation team concerned with the bulbar area, the speech therapist should be related integrally to the general therapeutic efforts. He must inevitably concern himself with matters of general bodily state, being aware of variations in quality of performance associated with intercurrent illness, fatigue or psychic distress.[1] He must be aware of the general principles and clinical problems of artificial respiration and of tracheotomy. He must be oriented to the general psychological situation of the patient and, as a therapist who is much associated with the patient and in an exceptionally effective interpersonal relation, he may contribute much to the patient's appropriate attitude toward disability and further efforts of therapy. Also, a particularly strategic circumstance is that of the variety of accessible therapeutic goals that the speech therapist may offer to the patient. The objectives of the therapist also may be modified as he participates in the general rehabilitation plan. Thus, he may continue his combined therapeutic efforts and personal support in the circumstance of comparatively little specific indication for speech therapy, but by reason of the patient's current psychic needs and the speech therapist's proven effectiveness in answering these needs.

It is highly probable that the vocalization exercises are of much value in rehabilitation of the larynx, which rehabilitation is of importance in improving pulmonary function.[11] The mechanisms of this contribution to pulmonary physiology and ventilatory action are several and include the achievements of cough, and of more appropriate regulation of pulmonary and general intrasomal pressure variations. It is our impression that the speech therapist has been of particular assistance in aiding the patient in adaptation to valving, progressive closure and eventual permanent closure and removal of the tracheostomy tube, possibly with its replacement by a tracheostoma obturator.

By integral participation in the rehabilitation team, the speech therapist is able to achieve a better orientation to the general somatic and psychic state of the patient, his respiratory adequacy and his general prospects for rehabilitation as a useful person in society. Without such awareness, the speech clinician may become impatient with his client or may attempt disproportionate encouragement, or otherwise confuse the patient by discrepant attitudes and advice. Also, of

course, it is presumed that the speech clinician is thoroughly aware of the patient's specific disabilities in the bulbar area. Without this awareness, his efforts are comparatively ineffectual and he may expend the time, attention, and energies of the patient to comparatively little profit.

SUMMARY

The extensive variety of disabilities of speech following poliomyelitis are classified and briefly described. The disabilities are commonly multiple and mutually exaggerating in their effect.

The role of the speech clinician in the general rehabilitation team is discussed, and it is emphasized that the speech clinician and his therapeutic measures provide a contribution greater than mere facilitation of communication, as the patient is thus given further personal support and as the procedures of speech rehabilitation facilitate recovery of other performance of the bulbar area.

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The authors are much indebted to Dr. David Dolowitz and Dr. George Kelemen for editorial assistance in preparation of successive drafts of this publication. Stenographic and medical illustration assistance were ably provided by Mrs. Dixie Olson and Mr. Julian Maack, respectively.

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V

RECONSTRUCTION METHODS FOLLOWING LARYNGOPHARYNGECTOMY

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The direct spread of a tumor by continuity and the extension by lymphatic channels in carcinoma of the hypopharynx is generally interpreted as condemning the larynx when adequate surgical management is undertaken. In the past, complete or partial laryngopharyngectomy has been indicated hesitantly because of sentimental thoughts of a patient deprived of normal phonation and deglutition. As a result, many otherwise salvageable patients have been relegated to palliative care consisting of inadequate surgery or debilitating radiation without any thoughts of complete eradication of malignant disease. With the present assurance that a satisfactorily functioning hypopharynx can be reconstructed, a more aggressive surgical attitude is encouraged. The removal of the hypopharynx and larynx, with or without a complete neck dissection in continuity, as may be indicated, is now undertaken with a reasonable assurance that a presentable, emotionally adjusted patient can be returned to a useful life.

The surgical management and the indications for neck dissection in continuity have been discussed elsewhere by ourselves[1,2] and others[3-5] and therefore only a brief summary is presented. Because of the rich lymphatic bed encountered at an early stage of growth, carcinoma of the hypopharynx frequently metastasizes to cervical lymph nodes early in the clinical course of the disease. Palpable lymph nodes demand a laryngopharyngectomy and complete unilateral neck dissection in continuity. When the origin of the primary tumor appears to be unilateral, experience has demonstrated that cervical metastases can be anticipated in about 50 per cent of cases.[6] Neck dissection in continuity is therefore advocated in anticipation of cervical node

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involvement. Where the hypopharyngeal tumor is essentially a midline lesion and there are no palpable cervical lymph nodes on either side, neck dissection is delayed until such time as a node may appear. If a node appears, complete neck dissection on the affected side is performed without delay.

The choice of incisions varies with the procedure planned. Where neck dissection is done, a horizontal H type of incision system is employed. The superior incision extends from the mastoid tip following skin lines to the level of the hyoid bone, crossing the midline to the anterior border of the opposite sternocleidomastoid muscle. The inferior incision extends from the anterior border of the trapezius muscle at the level of the clavicle, curves superiorly, then inferiorly, to cross the trachea horizontally above the sternal notch, and across the midline to the anterior border of the sternocleidomastoid muscle. A paramedian vertical incision joins the two horizontal incisions.

For laryngopharyngectomy without neck dissection, the incisions must permit reconstruction and not interfere with possible access to either side for probable subsequent neck dissection. Furthermore, the incisions employed should not interrupt normal lymphatic channels if bizarre metastatic spread is to be avoided. The most suitable incision to meet the requirements of these specifications is a "trap-door" type. A horizontal incision over the hyoid and above the sternal notch are joined by a vertical paramedian incision. In addition to affording good exposure, the closure with such a flap following removal of the larynx and pharynx affords a good skin covering for the bared prevertebral area.

In addition to the two types of incision, there are two possible sites for the formation of the cervical esophageal stoma. It may be placed in the midline superior to the tracheostomy if there is sufficient remaining esophageal mucosa. Or, where the transection has been made at a relatively low level and the tracheostomy would prevent midline placement, the cervical esophageal stoma is formed laterally. Since placing it immediately lateral to the tracheostomy creates healing and drainage problems, it is brought further lateral. This is accomplished with ease, and it may be placed as far lateral as the site between the sternal and clavicular heads of the sternocleidomastoid muscle or medial to the sternal head.

The problem for reconstruction is not altered greatly by the forms of incision, as in each type the midline area is covered by a closed

"door," a vertical paramedian scar on one side and a broad unscarred pedicle on the other. However, the medial or lateral placement of the esophagostomy does pose individual problems in planning the subsequent reconstruction.

The reconstruction of the hypopharyngeal-esophageal tube is undertaken as soon as the original wound healing is complete. It would seem to be feasible to initiate flap elevation at the time of initial resection, but it is generally inadvisable to prolong the procedure at this time. Experience seems to indicate that there are some advantages in delaying the onset of repair until initial healing is well along in order to permit tissues to return to normal consistency, free from edema, cellulitis and contraction. The skin also seems to become adapted to some bathing by salivary secretions and develop some degree of immunity to organisms usually present. The patient is maintained in good nutritional balance by liquid and soft foods and dietary supplements by employment of the esophageal stoma. The surgical reconstructive phase may be instituted as early as two weeks following resection.

Reconstruction of organs of some thickness that require an inner and an outer epithelial surface requires the use of skin flaps that include skin and subcutaneous tissue detached from its underlying support, but still partially connected at its periphery on its base as the source of its blood supply. The Indian method of flap rotation is employed and the principle of delayed transfer is used in the manner re-emphasized and popularized by Blair.[7] They are nontubed and single pedicled, the flaps being cut just below the subcutaneous fatty layer since the blood vessels which nourish the flap lie above and in the layer of subcutaneous tissue.

Selection of the donor site is governed by several considerations. It must be in the immediate neighborhood since the flap can be shifted only so far as its pedicle or basal attachment will allow. In outlining the flap, an area approximately 40 per cent greater than the apparent need is taken and width to length ratio of three to eight is maintained. Further, the site must be nonhairbearing; it must also be unscarred since scar tissue across a flap will obliterate the distal blood supply.

The major difference in technique for the two sites of the cervical esophageal stoma rests in the fact that with midline placement, the skin of the neck is tubed and covered by a skin flap (Figs. 1 through 6); with lateral placement of the cervical esophageal stoma, the flap is tubed and covered by the skin of the neck (Figs. 7 through 12).

When the site of the skin flap is selected and its size determined, the first stage of flap preparation consists of an "L" incision through skin and subcutaneous fatty tissues, followed by undermining and replacement. The next step, seven to ten days later, consists of reincising the "L" and completing the second long incision, undermining the flap to its attached base and replacing it for approximately 21 days.

The details of each approach to the problem are presented in the accompanying illustrations and are described in their accompanying legends.

The nasogastric tube is removed from the reconstructed food passages in from 8 to 14 days. It can be replaced if fistulas occur, but in the absence of fistulization, the patient is started on fluids by mouth. The nasogastric tube is removed and the patient is advanced from soft to solid foods as rapidly as he can accept them, since food is an excellent physiological dilator at this stage of healing.

The reconstructive methods described have been used with satisfactory results in ten patients. The types of their lesions have been extensive, bulky carcinomas of the hypopharynx of such degree as to prevent any salvage of mucosa if resection was to adequately deal with the tumor. Representative cases are presented to illustrate the techniques which have been discussed.

REPORT OF CASES

CASE 1. A 53 year old female with a postcricoid carcinoma underwent laryngopharyngectomy. Sufficient hypopharynx remained to effect primary closure. Twenty-eight months later she complained of an inability to swallow. An exophytic persistence or recurrence of the tumor inferior to the original site was detected and a further excision was made through a trap door type of incision. A cervical esophageal stoma was formed in the midline. Reconstruction was carried out in the stages as outlined in Figures 1 through 6 and the final outcome with a functioning food passage is shown in Figure 13.

CASE 2. A 66 year old man was observed to have a bulky malignant tumor involving the posterior commissure, postcricoid area and embracing the anterior two-thirds of the circumference of the cervical esophagus. Since there were no palpable cervical nodes, laryngopharyngectomy was performed without neck dissection through a trap-door incision. Because of extension of the growth into the cervical esophagus, a low, lateral placement of the esophageal stoma was

required. Reconstruction was completed as shown in Figures 7 through 12 and was functional in three months after the initial excision. Five months after the primary surgery, lymph node involvement was detected in the left anterior triangle and a left complete neck dissection was carried out. Reconstruction had not complicated the surgical neck field, nor did the neck dissection alter the reconstruction (Fig. 14). The patient remained well for three years and succumbed to an adenocarcinoma of the stomach.

The reconstruction methods described can be performed where neck dissection has been carried out as a monobloc procedure without interference to or from such flaps. This is the more common experience. But also significant is the fact that the incisions of the reconstruction do not interfere with future neck dissection (as in Case 2) on either side, nor does subsequent neck dissection disrupt the re-established food passage.

The major complications of management of this type of patient consist of flap failures, postreconstruction fistula and recurrence or persistence of disease. Flap failures are generally the result of hemorrhage or cellulitis with eventual slough. If the flap is properly prepared by carrying its depth to the pectoral fascia, properly elevated and delayed, this complication is reduced to an extremely small number and was not experienced in the present series.

Indirect, circuitous fistulas generally close without specific care, whereas direct fistulas may require secondary closure.

The problem of recurrence or persistence of disease is, of course, of primary concern in surgical management of malignant tumors. Adequate excision of the primary growth and its paths of potential lymphatic spread as a bloc has made considerable improvement in the survival results which have been experienced. The confidence in a truly satisfactory reconstruction method has, in turn, encouraged adequate resection without undue concern over failure to return a presentable functioning patient to society. Armed with a successful closure method, the faith in extensive surgery results in adequate excision.

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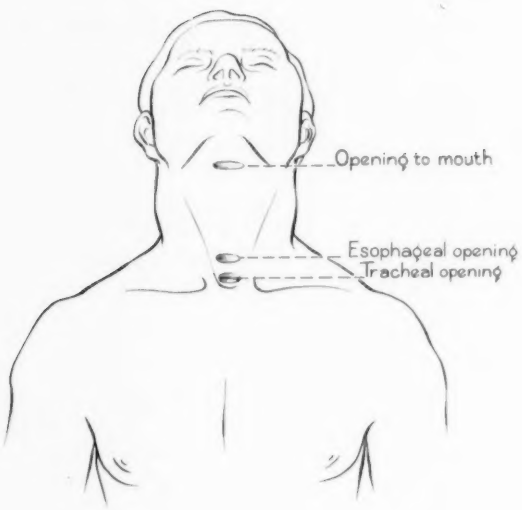

Fig. 1.—Postoperative condition with midline placement of the cervical esophageal stoma superior to the permanent tracheal stoma. The continuity of the food channel is re-established by forming a tube, skin side in, connecting the mouth and esophageal stoma. The tube and bared skin areas are then covered by a pedicle skin flap from the chest. The size of the pedicle flap is estimated and the site is selected as described in the text.

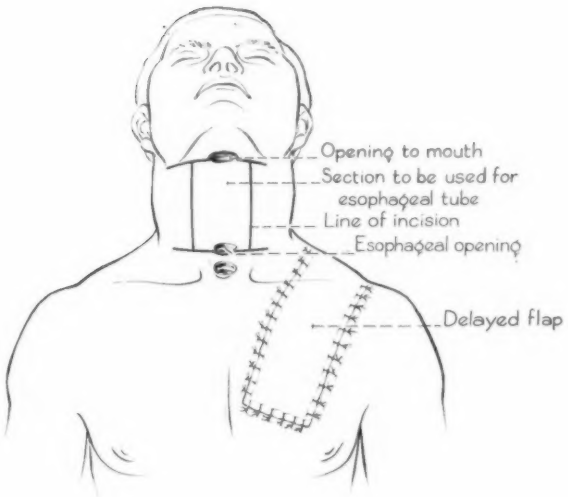

Fig. 2.—The acromioclavicular delayed flap has been prepared after estimating the area required and the distance to be traversed. An area 40 per cent greater than the apparent need is taken and a width to length ratio of three to eight is maintained. The first elevation is made by an "L" shaped incision, undermining and replacing it for seven to ten days. The flap is again elevated, this time completing the second long incision and replacing it for 21 days. This is the status indicated above, when the actual reconstruction is begun. The initial neck incisions are reopened to the extent that the mucocutaneous margin of the pharyngeal stoma is opened in its anterior one-half, separating skin and mucosa. Intervening scar tissue is removed so that clean, accurately defined edges of skin are present. The esophageal stoma is opened and trimmed in a similar way.

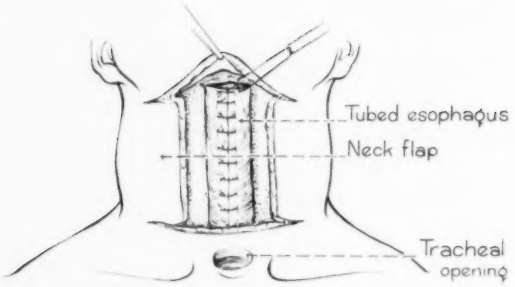

Fig. 3.—A tube is formed by making two vertical paramedian incisions approximately 2.0 cm from each lateral extent of the pharyngeal opening to a similar distance lateral to the esophageal opening. The flaps thus formed are undermined medially. The flaps are rotated to form a tube, skin surface inside. The freshened esophageal mucosa is sutured to the inferior margin of the tube using absorbable sutures with knots tied so as to be inside the tube. The freshened edge of the pharyngeal mucosa is attached to the upper end of the tube in a similar manner.

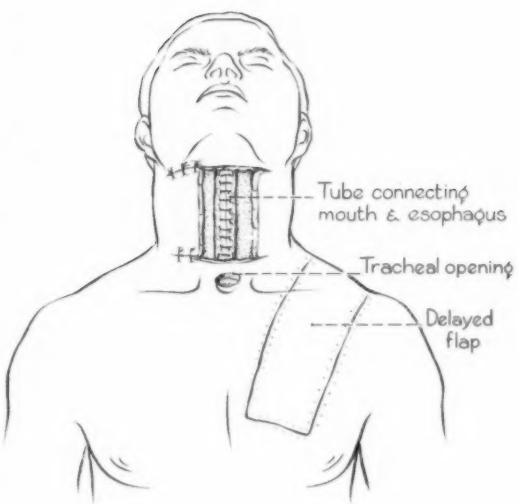

Fig. 4.—Thus, the connection between pharynx and esophagus is formed, raw surfaces remaining on the anterior surface of the newly formed tube as well as lateral to it on each side. The acromioclavicular delayed flap having been previously prepared, is raised.

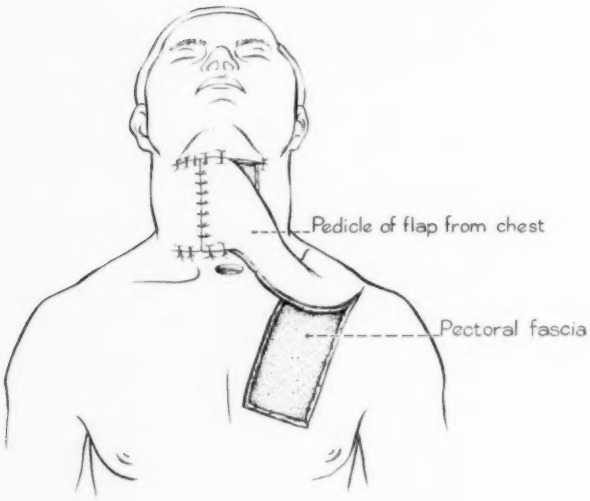

Fig. 5.—The raised flap is moved into position and the distal edge is sutured to the skin edge of the neck defect on the side opposite the flap. This vertical suture line does not correspond with the vertical suture line of the tube. The flap is also sutured to the horizontal incisions which were reopened in detaching the stomata at the outset of the procedure. The pedicle is left attached for 21 days. A nasogastric feeding tube is guided through the newly formed tube. This may be left in for 8 to 14 days and is removed if there are no fistulas present. During the waiting period the flap becomes attached to the bared areas and the raw surface of the newly formed tube; the attachment extends as far as the cutaneous margin of the opposite side. The steps illustrated in Figures 3, 4 and 5 are carried out at one stage.

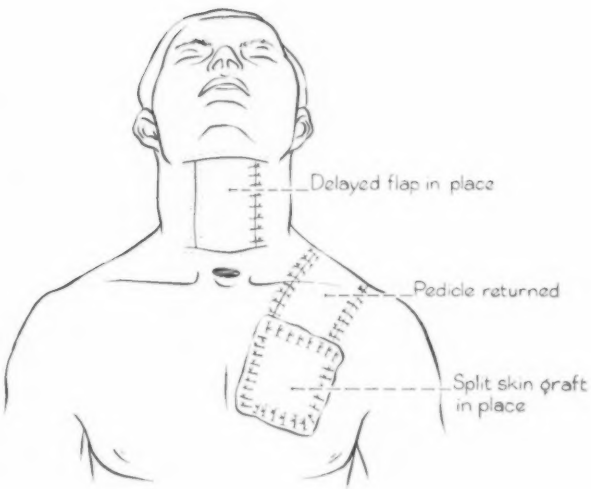

Fig. 6.—After the waiting period of 21 days, the pedicle is severed. The scarred edges of the flap are trimmed and the remaining pedicle of the flap is returned to its original bed. There is an area of granulation left uncovered by this return. The edges of the granular defect are trimmed away. A thick split graft is removed from a convenient donor area, usually the thigh and is placed over the granulating area of the donor site. A pressure dressing is applied and the split graft site is similarly dressed with petrolatum gauze.

The newly severed edge of the pedicle flap is fitted into place. This requires removal of scar from the skin edge to which it is to be sutured, as well as undermining. Interrupted sutures are then placed and a nasogastric feeding tube is inserted. The tube is permitted to remain for approximately eight days.

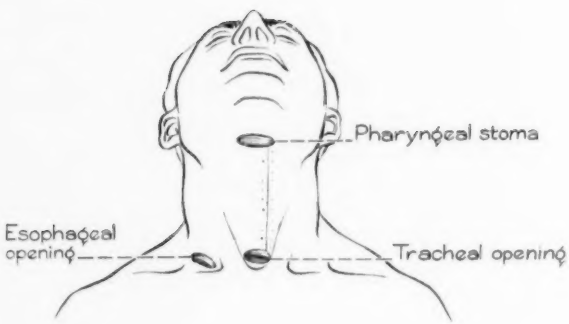

Fig. 7.—Postoperative condition with lateral placement of the cervical esophageal stoma. In this stage the food passage is reformed by creating a tube from a pedicle flap and covered by the skin of the neck.

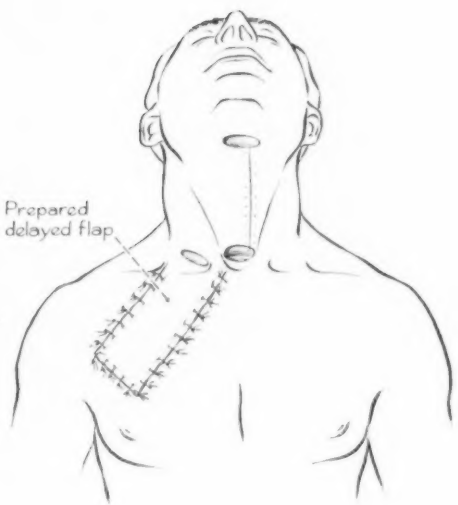

Fig. 8.—The delayed pedicle flap is prepared as previously described. Its blood supply is assured by raising and replacing. It is made approximately one-third larger than the measurements from the upper to lower stoma, and also one-third wider than the anticipated needs, bearing in mind that a final diameter of approximately 2.0 cm is desirable for the reconstructed tube. The average flap is approximately 8.0 by 20.0 cm.

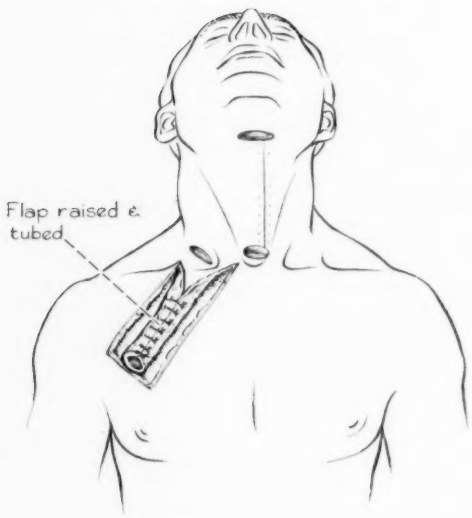

Fig. 9.—After the flap has been prepared, it is raised and formed into a tube, with the skin side inside, by suturing the two lateral edges.

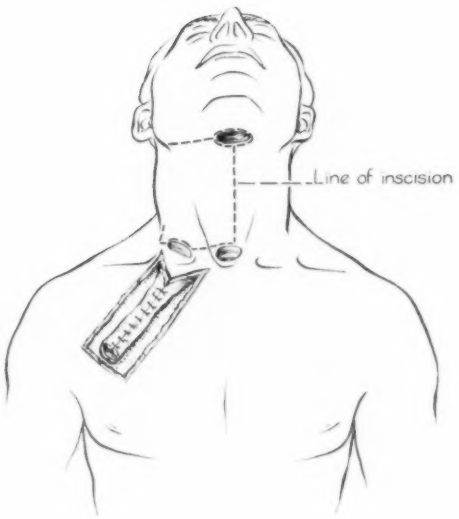

Fig. 10.—The neck incisions are reopened and undermined. The undermining is carried sufficiently lateral to permit burying of the newly formed tube. The posterior half of the circumference of the esophageal stoma is freshened, as in the entire circumference of the pharyngeal stoma in order to present fresh, well-defined edges.

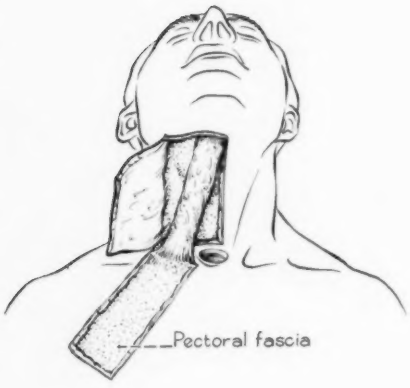

Fig. 11.—The proximal end of the newly formed tube is then attached to the posterior aspect of the esophageal stoma. The distal end of the tube is brought up to the pharynx and the mucosa attached to the distal skin circumference. The prepared esophagus now connects the stomas, re-establishing the continuity of the food passage. It lies in the subcutaneous layer.

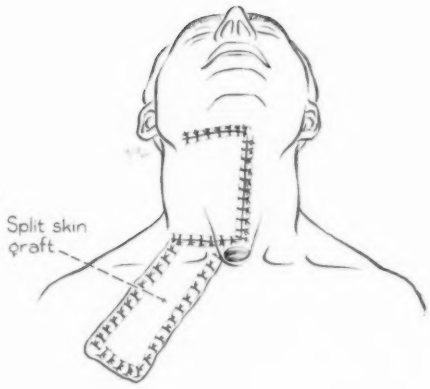

Fig. 12.—The reflected skin flaps of the neck incisions are brought together and resutured over their previous sites and also over the subcutaneous surface of the newly formed tube. A nasogastric tube is inserted for feeding. A split skin graft is placed over the donor area of the original pedicle flap. A pressure dressing is applied.

Figures 9 through 12 are carried out at one operation.

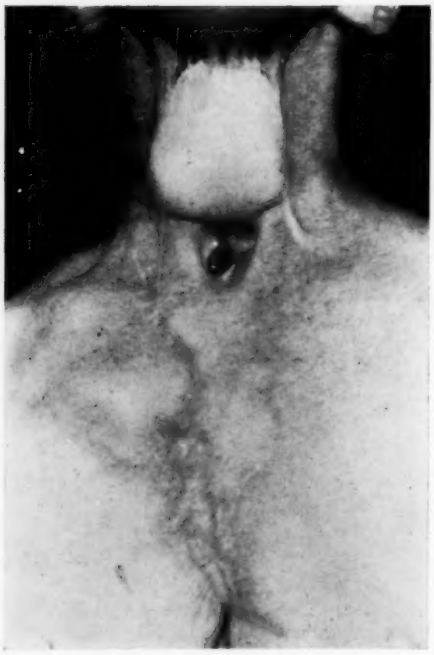

Fig. 13.—The reconstruction following laryngopharyngectomy has been completed in Case 1 by the method presented in Figures 1 through 6. The lateral neck areas remain unscarred and present an unaltered area should the need arise for neck dissection.

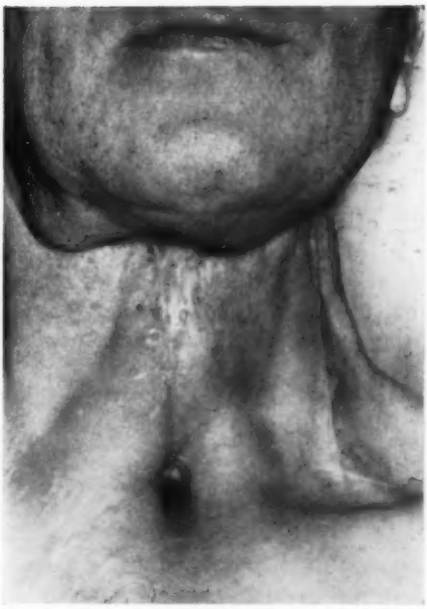

Fig. 14.—The completed reconstruction of Case 2 by the method described in Figures 7 through 12. Subsequent to completion of the reconstruction, left neck dissection was required.

VI

PHYSICO-CHEMICAL PROCESS IN THE HAIR CELLS OF THE ORGAN OF CORTI

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The hair cells of the organ of Corti require a continuous supply of energy in order to maintain the integrity of both structure and function of these cells. It is well accepted that the cochlear microphonics, action potential and dc potential are maintained by metabolic energy, because these potentials are depressed during oxygen lack.[1]

The toxic effect of dihydrostreptomycin and quinine on the cochlea has been known for a long time.[2-4] Moreover the permanent acoustic trauma could be explained in animal experiments by morphological changes or depression of the electrical activity of the hair cells of the cochlea. In these well-known experiments the stimuli used to cause the microscopic changes of the organ of Corti and the reduction of cochlear microphonics were those of high intensity of long duration, which were not usually encountered in our environment.

The auditory fatigue is accepted as the pre-stage of the permanent acoustic damage of the cochlea, in which the hair cell shows apparently normal structure by stain technique heretofore in use. The main purpose of this paper is to reveal the correlation between the histochemical changes and the electrical activity of the hair cells, when the metabolism of the cochlea was slightly disturbed by relatively loud sound of short duration or by the toxic effects of quinine or dihydrostreptomycin.

METHOD

Carefully selected healthy guinea pigs were treated with daily subcutaneous injections of dihydrostreptomycin sulfate in amounts

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equivalent to 200 mg of dihydrostreptomycin base per kg of body weight for a period up to 60 days. Total quantity of dihydrostreptomycin sulfate reached 6 to 10 gr. One hundred mg of quinine per kg of body weight was injected into another series of guinea pigs and we administered total quantities of 500 mg to 2 gr.

Sound stimuli for exposure were accomplished by a loud speaker driven by an audiometer in conjunction with a power amplifier. A plastic tube was connected to a loud speaker and the opening of the tube was inserted into the auditory canal of the animal. Sound level was measured by a sound level meter. The tube connected to the loud speaker was placed to the microphone of the sound level meter and the sound level was determined.

Cochlear microphonics was recorded from a pair of differential electrodes. The tip of the silver electrode was approximately 30 microns.

Guinea pigs, anesthetized with urethane (1 gr/kg), were used for the experiment. Tracheotomy was performed and an incision was made through the skin in the mandibular region. The operative technique of exposing the auditory bulla similar to that described by Tasaki and Fernandez[5] was used. The bulla was opened with a dental drill to expose the cochlea. The head of the animal was fixed on the operating table with small clumps. Care was taken to avoid any injury to the drum membrane and the ossicular chain. Two small holes were made at points in directly opposite positions across the cochlear duct and close to the round window within the basal turn, through which electrodes were inserted into the scala vestibuli and the scala tympani with a micromanipulator under the dissecting microscope. The arrangement of apparatus is shown in Figure 1. The reference electrode was placed in the neck muscles. The electrical responses were led to the differential preamplifier and observed or photographed on an oscilloscope.

The sound stimuli we employed were continuous pure tones and also a brief click. A single click was used to avoid interference due to successive stimulation. Figure 2 shows the wave shape of the stimulus recorded with a microphone which is placed in the position occupied by the head of the animal.

Immediately after the cochlear microphonics measurements were finished, the carotids were cannulated and the head perfused from a

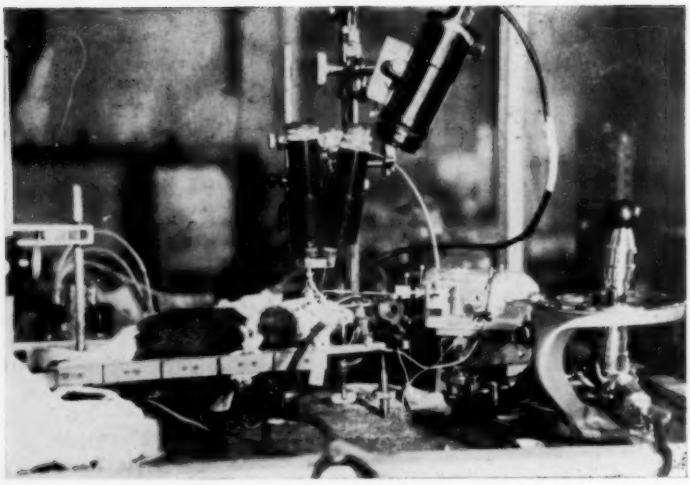

Fig. 1.—Arrangement of the apparatus for the measurement of the cochlear microphonics.

reservoir bottle first with Ringer's solution to wash out the blood and then with Carnoy's solution to fix the tissue. The ear was removed with great caution, the adjacent bone to the cochlea was removed and the cochlea was then immersed in Carnoy's solution. The beam of light was focused on the specimen and the outer bony shell was taken off with a small hook under the binocular dissecting microscope with a magnification of about 20 times. By removing the bony shell from the apex to the first turn, the stria vascularis and the osseous spiral lamina were visible. Using a small knife and scissors, the preparation was removed to another glass, containing Carnoy's solution; this included the osseous lamino, the organ of Corti and the spiral ligament. In order to ascertain the distribution of ribodesose nucleotides in the hair cells, the Feulgen nuclear reaction was performed.[6] The specimen was subjected to the hydrolysis procedure of the Feulgen reaction (10 minutes in 1N HCl at 60° C), immersed in Schiff's solution for one hour and next in a solution of sulphurous acid. After washing for one minute, the preparation was dehydrated and mounted.

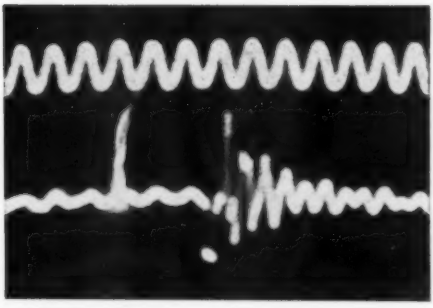

Fig. 2.—The wave form of the click stimulus recorded by a microphone, the intensity of which is approximately 90 db above the human hearing threshold.

RESULTS

Experiment with Non-Treated Guinea Pigs. The auditory fatigue is a reversible condition of the auditory sense organ, in which the effect of an acoustic stimulation becomes diminished after the termination of stimulation. The amount of fatigue increases with the intensity of the tone employed and its duration. In the present series of experiments we used pure tone of 4000 cps of approximately 90 db above the human hearing threshold.

In order to demonstrate the recovery from the temporary decrease in the cochlear microphonics the short duration fatigue was used. The duration of exposure tone was from 30 to 60 seconds. Figure 3 demonstrates the cochlear microphonics to click in the basal turn in which the figures show the period after the termination of exposing tone. The recovery began rapidly during the first seconds and progressed slowly. At the conclusion of the cochlear test, the temporal bones were ready for histochemical preparation and study. Under low-power magnification, the inner and the outer hair cells of the organ of Corti were distributed regularly; by the Feulgen reaction, cell nuclei of the inner hair cells form a single row and those of the outer hair cells, three spiral rows as demonstrated in Figure 4. It is remarkable that the arrangement of these nuclei of the hair cells is not irregular and the magnitude of nuclei does not show any great variation.

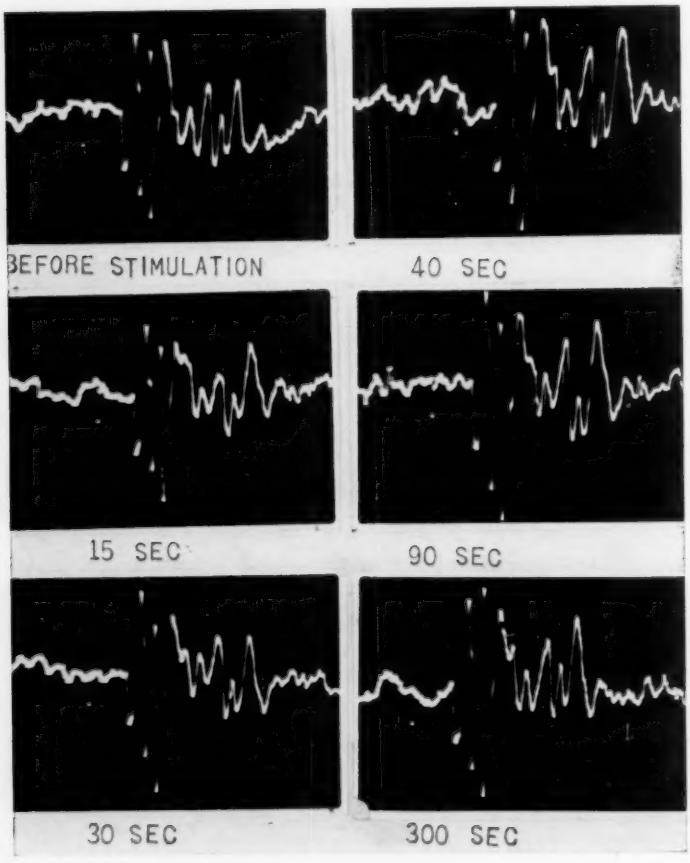

Fig. 3.—Recovery of the cochlear microphonics to click after exposure. Normal guinea pig 131. The exposing tone employed is 4000 cps, 90 db lasting 60 seconds. The recovery begins rapidly and the overshoot is observed after the complete recovery in this case.

Those parts of the hair cells which are positive to the Feulgen reaction are situated solely within the nucleus, and the cytoplasma gives a completely negative result. The distribution of the Feulgen positive substance was found to be normally as follows: the nucleolus gives a positive reaction and coarse clumps of chromatin are distributed irregularly around nucleolar border.

Experiment with Quinine-Treated Guinea Pigs. Healthy adult guinea pigs were treated with daily subcutaneous injection of quinine chloride in an amount equivalent to 100 mg of quinine per kg of body weight for a period up to 15 days.

We examined the sensitivity of the cochlear microphonics in three guinea pigs poisoned by quinine. Care was taken not to change electrical conditions, because the amount of change varies greatly with the circumstance, such as moisture of the cochlear surface or overflow of the perilymph through the electrode inserting hole. The measurements showed that the amount of quinine caused no depression in acuity for tones from 250 to 8000 cps. A moderate increase in quinine reduced the acuity for high tones without affecting the acuity for low tones.

The auditory fatigue test on the cochlear microphonics was carried out in a group of the guinea pigs showing no decrease of acuity for cochlear microphonics for testing tones. Pure tone of 4000 cps of 90 db above threshold lasting five minutes was used as the exposing tone. A click was generated every five seconds after the termination of the exposing tone. The recovery of temporary depression of the cochlear microphonics was observed. Figure 5 shows the temporary depression of the cochlear microphonics due to exposing the intense sound in a quinine-treated guinea pig. The decrease of the cochlear microphonics developed more rapidly than those observed in normal control animals, but the recovery began rapidly and the temporary reduction of the cochlear microphonics was completely recovered in all cases. Among all experimental cases the individual variations were observed when using large intensities.

After complete recovery was observed in the cochlear microphonics, the guinea pigs were prepared in order to perform the Feulgen nuclear reaction. The preparation showed that the nuclei of the outer hair cells were regularly distributed and the shape of the nucleus was either round or oval. Compared with those observed in control animals, these nuclei varied in size. The Feulgen reaction positive sub-

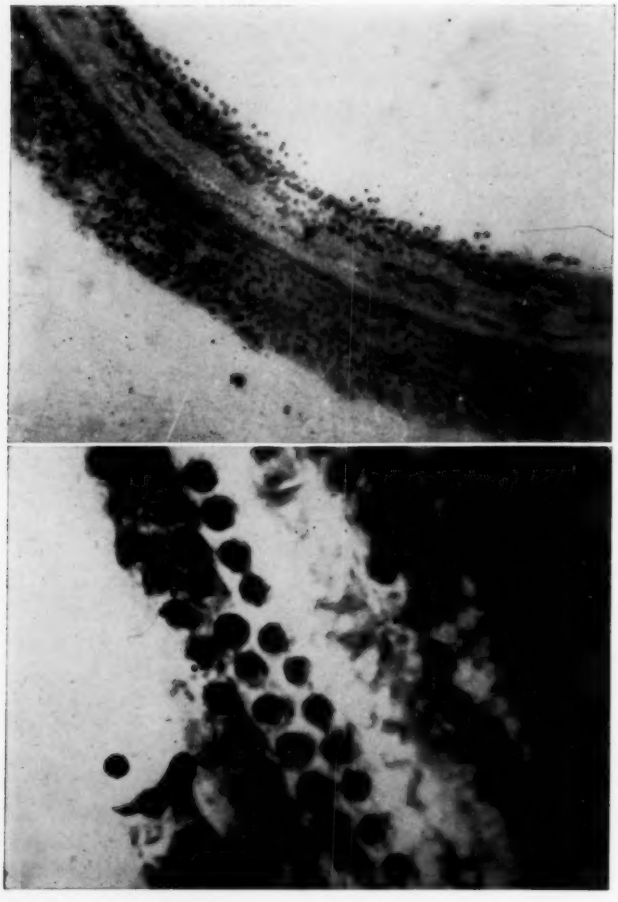

Fig. 4.—The Feulgen reaction in normal hair cells. The outer hair cell nuclei form three spiral rows and the inner hair cell nuclei a single row. They arrange regularly and show no variation in size and shape. The nucleolus as a whole gives a positive reaction and coarse clumps of chromatin are distributed around the nucleolar border.

stance was mainly located around the nuclear border and was fairly granular with occasional prominent clumps as shown in Figure 6. Though the chromatin content was somewhat irregular, the nuclear border did not condense and was smooth.

Experiment with Dihydrostreptomycin - Treated Guinea Pigs. Dihydrostreptomycin in the larger dosage employed in the series of animals caused reduction of the cochlear microphonics as already pointed out in some other publications. The cochlear microphonics was affected more or less in all audible frequencies from 250 to 8000 cps or especially in high frequencies up to 4000 cps. Even the application of a tone of moderate intensity caused a severe auditory fatigue in a group of dihydrostreptomycin-poisoned guinea pigs. Figure 7 shows an example of marked depression of the cochlear microphonics for click after exposure to a tone of 4000 cps 90 db lasting 60 seconds. This case showed slight decrease in sensitivity of the cochlear microphonics for high tones and no depression for low and middle tones. Recovery began slowly and progressed more and more slowly up to 30 or 60 minutes. The recovery process showed the individual variation, and the transition into permanent depression of the cochlear microphonics was observed in some cases.

The Feulgen reaction was performed in a series of guinea pigs in which there had been no evidence of cumulative injury by evaluating the cochlear microphonics. The row of the hair cells, as demonstrated in Figure 8, was distributed with remarkable irregularity, and most of the outer hair cell nuclei illustrated no less remarkable variations both in size and in shape. Noticeable increase in Feulgen reaction positive substance in nuclei and compact appearance was seen which is characteristic for pyknosis. Contrary to these findings, other nuclei showed a marked increase in size, and the nuclear border was not clearly defined. Fine particles of the Feulgen reaction positive substance were scattered eccentrically, as was generally the case with karyorrhexis. In careful observation we could find absence of cells in the irregular row of the outer hair cells. The Feulgen reaction was completely negative in nucleus which was of karyolytic quality.

Guinea Pigs Subjected to Acoustic Stimuli. A group of healthy guinea pigs was exposed by pure tone of 4000 cps, intensity of which was approximately 90 phon, in front of the tympanic membrane of the experimental ear. The duration of the exposing tone was varied from 30 to 40 minutes. The cochlear microphonics for the click produced by impulse generator was recorded successively before and after

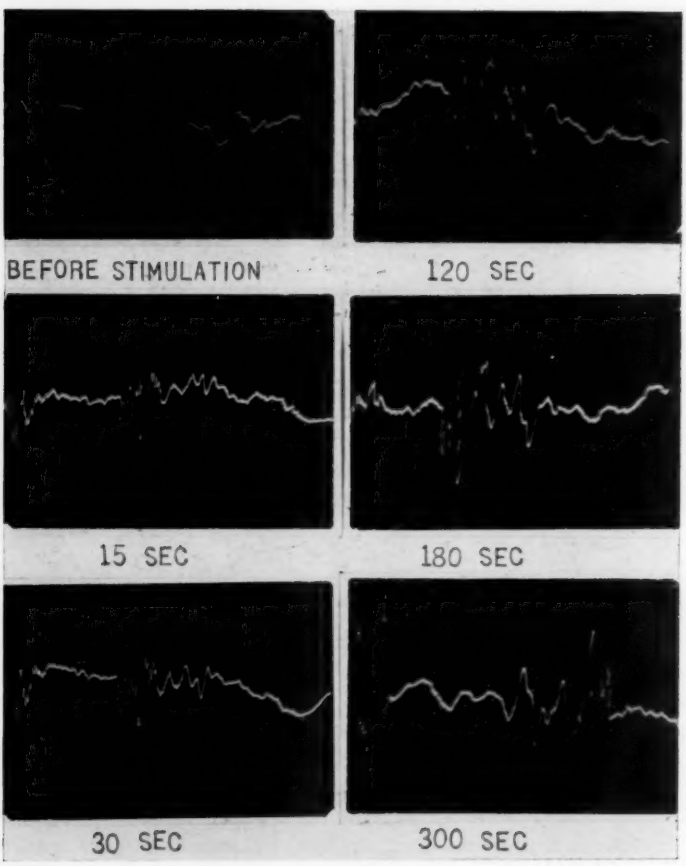

Fig. 5.—Recovery of the cochlear microphonics to click after exposure in quinine intoxicated guinea pig. Guinea pig 150; quinine 500 mg. Exposing tone employed is the same as that in Figure 3, but the duration is 5 minutes. Recovery begins rapidly but more slowly than those observed in normal guinea pigs. The complete recovery is observed in all cases.

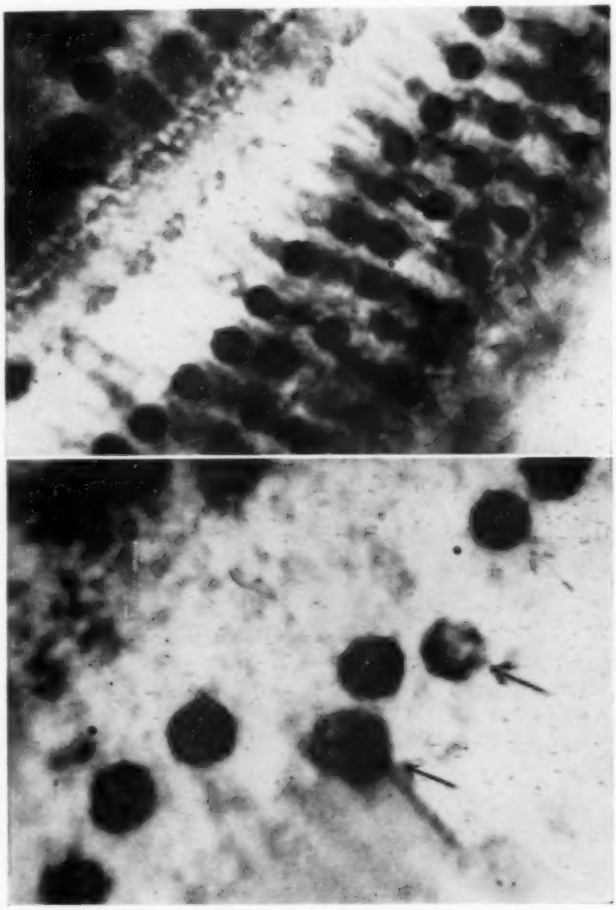

Fig. 6.—The Feulgen reaction of the hair cells in guinea pigs intoxicated by quinine. The arrangement of the outer hair cell nuclei is irregular and the chromatin is located around the nuclear border and is fairly granular with occasional prominent clumps, as illustrated under high power magnification.

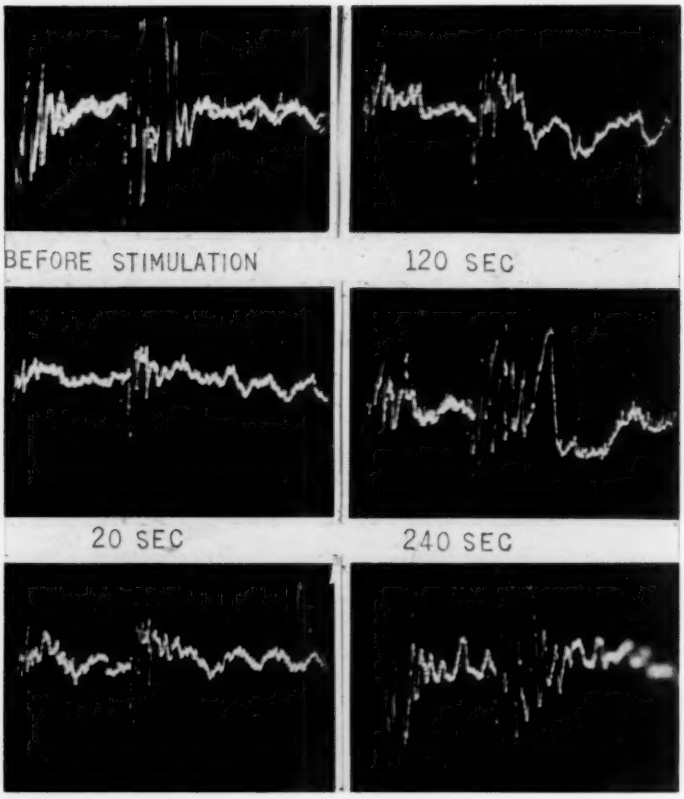

Fig. 7.—Recovery of the cochlear microphonics to click after exposure. Guinea pig 45; DHSM 49g. Exposing tone employed is the same as that in Figure 3. The depression of the cochlear microphonics caused by exposure is remarkable and the recovery process is markedly slowly.

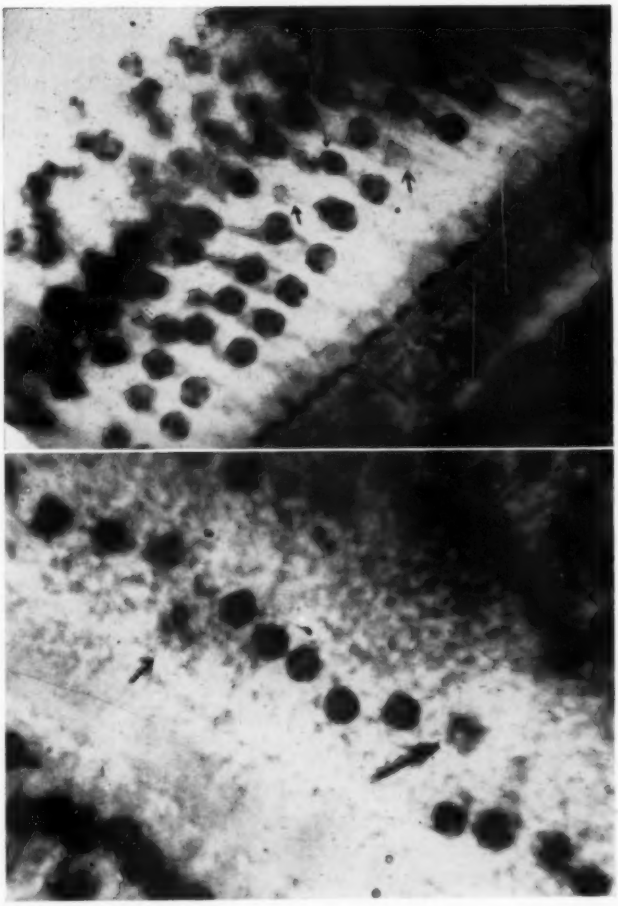

Fig. 8.—Feulgen reaction of the hair cells in dihydrostreptomycin toxicated guinea pigs. Cell nuclei show a large variation in size and shape and irregular distribution. Pyknosis, karyolexis and karyolysis are found in the outer hair cell nuclei.

the ear had been exposed to stimulation. Care was taken to minimize displacement of the animal's head and to maintain the respiration of the animal at a practically unimpaired rate during the recording of the cochlear microphonics.

A certain amount of time was necessary to fatigue the healthy ear by means of the exposing tone employed in this investigation, because the recovery process took place more rapidly than the interval between upset of fatigue tone and click. Figure 9 shows the decrease of the cochlear microphonics to click due to the fatigue effect during the stimulation tone described above. The cochlear microphonics were decreased slowly during the first ten minutes. The temporary depression of the cochlear microphonics was prominent after ten minutes and developed rapidly. The results were taken some 40 minutes after the fatigue tone had ceased, showing complete recovery from the reduced sensitivity of the cochlear microphonics, and no evidence of permanent injury was to be noticed.

Other series of animals were fatigued by the same exposing stimulation, and were prepared to perform the Feulgen reaction immediately after the exposing tone ceased. As shown in Figure 10, most of hair cell nuclei in the outer hair cell row demonstrated the prominent pyknotic character. On the other hand, karyorrhexis was observed in some of the outer hair cell nuclei. Under the low-power magnification the row of hair cells, especially that of outer hair cells, was irregularly distributed and the cell nuclei varied in size.

COMMENT

The nature of the ototoxicity of dihydrostreptomycin has been a matter for active investigation and discussion. These observations immediately arouse the interest of otologists, because the toxicity of this antibiotic seems to be more highly specific than that of any other drug known to affect cochlear function. For this reason dihydrostreptomycin might be expected to furnish a useful experimental tool for the investigation of the cochlear function. It has been known for a long time that quinine produced hearing impairment by its direct toxic effect on cell cytoplasma, producing degeneration of the hair cells of the organ of Corti.

The point of toxic attack of dihydrostreptomycin and quinine in the aural region has not been definitely determined, though the question of the nature of the action has received particular attention and these studies have been pursued in many laboratories and hospitals.

In recent animal experiments concerning these toxic effects much attention was given to the relation between degree and form of functional loss on the one hand and the extent and localization of the histological changes on the other. These previous observations have thus shown that histologically and functionally demonstrable change occurs in the hair cells in dihydrostreptomycin or quinine intoxicated animals. But they have not enlightened us regarding the reaction of the hair cells nor as to recovery time if the experiment is discontinued at an early stage after the permanent impairment of function which may be presumed to precede the marked changes observed.

The cochlear microphonics are linked directly to the energy metabolism of the hair cells of the organ of Corti, as described in our publication[7] and others.[8] Overstimulation results in the diminution of cochlear microphonics in guinea pigs and a subsequent histological study reveals a cochlea that is normal in appearance. The earliest stage of reversible fatigue after exposure to sound cannot be disclosed by visual examination. Modern cellular research has made it possible to follow a certain functional process in the individual cell.[9]

It may be safely assumed that a slight disturbance of cell metabolism in the cochlea might be accelerated by moderate stimulation. The recovery time is defined as the interval from the termination of the fatiguing tone to the complete recovery of cochlear microphonics. This time is an index of speed of the physico-chemical reconstruction of cells as stated by Fernandez.[1] Figure 11 illustrates the recovery process for cochlear microphonics in an intoxicated animal by dihydrostreptomycin or quinine. It was 20 to 40 seconds in normal healthy ears. These responses show signs of recovery as rapidly after 30 seconds of stimulation as after one minute and similarly, as rapidly with low as with high intensity of click. This indicates that there is no sign of fatigue due to repeated click stimulation.

However, in a group of dihydrostreptomycin or quinine treated animals the recovery time was prolonged, as illustrated in Figure 11. Thus it is apparent that there is a marked difference in the speed of physico-chemical reconstruction of hair cells between normal and intoxicated animals by dihydrostreptomycin or quinine.

Tonndorf reported that the production of cochlear microphonics was shown to depend upon the oxygen metabolism of the organ of Corti, but only in that the degree of such dependence varied with sound exposure. He concluded that the effect of moderate oxygen depriva-

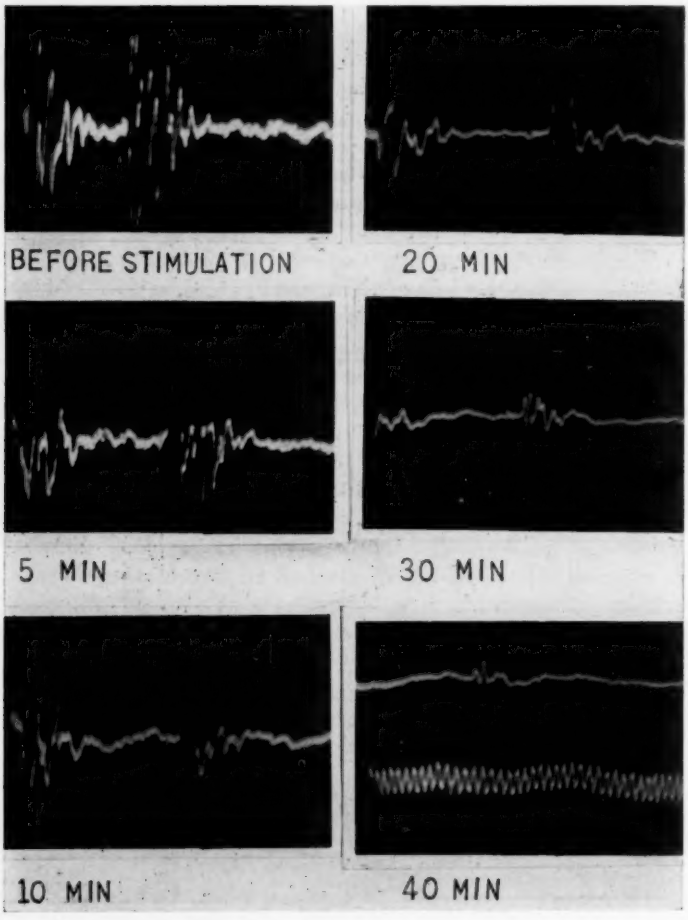

Fig. 9.—Depression of the cochlear microphonics to click caused by fatigue during stimulating tone of 4000 cps, 90 db, lasting 40 minutes. The temporary depression of the cochlear microphonics is prominent after ten minutes. Lower trace in the last figure is time scale of 1000 cps.

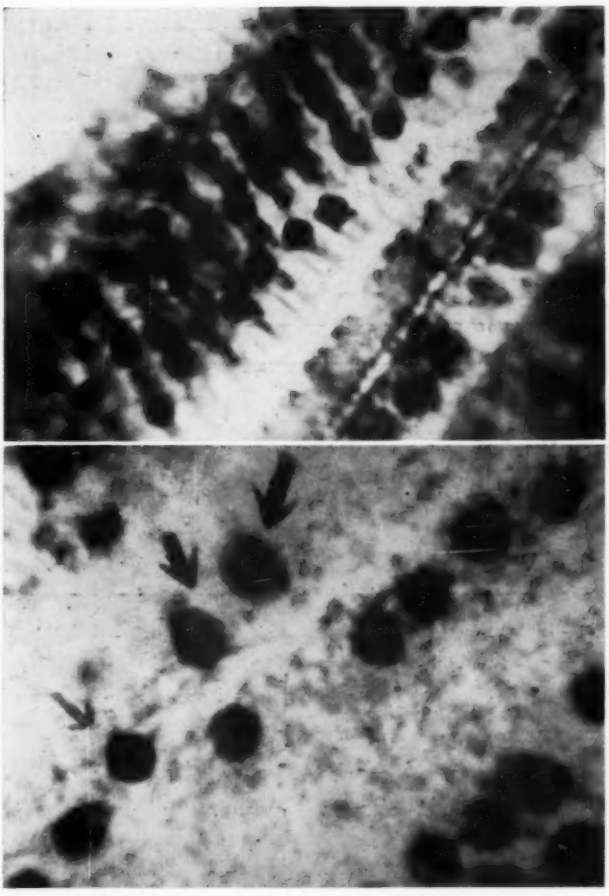

Fig. 10.—The Feulgen reaction in hair cells in overstimulated guinea pigs. Most of hair cell nuclei demonstrate pyknosis and are irregularly distributed.

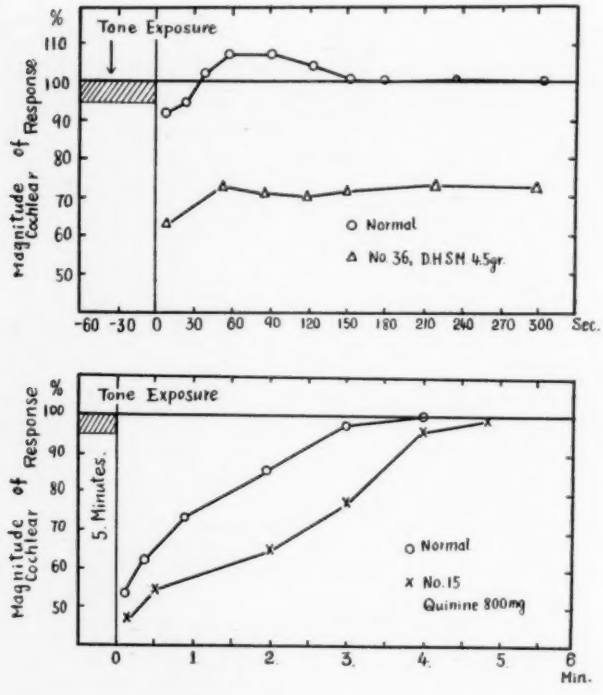

Fig. 11.—The recovery curve of the cochlear microphonics after the exposure to sound of 4000 cps, 90 db, in normal and intoxicated guinea pigs.

tion upon cochlear microphonics is most marked during exposure to sound and less so during recovery from sound exposure. It is concluded that it is responsible for the fatigue that gives manifestation to the latent physico-chemical disturbance in the hair cells.

The nucleus is the important organ of the cell for the formation of protein. The protein molecule-nucleic acid group is confirmed as the simplest carrier of life.[6] Wever stated the earliest stage of overstimulation might consist of biochemical changes, perhaps something like loss of nucleic acid and other proteins that Hamberger and Hyden by special methods have disclosed in the cochlear ganglion cells after overstimulation.

The Feulgen reaction is regarded as the most suitable to examine the function of the cellular protein forming system. This reaction gives positive results in the presence of ribodesose nucleotides and it guides the processes which constitute its function within the cells.

Both after acoustic fatigue and by intoxication of dihydrostreptomycin or quinine extensive changes could be observed in the cochlear hair cells of guinea pigs. These findings can be seen in the nucleus for the production of cellular protein. Hamberger and Hyden[6] reported that the changes in the more intensive stimulation consisted in a reduction of the content of nucleotides and proteins within a circumscribed part of the cytoplasma in the cochlear ganglion.

In the hair cells of guinea pigs which had not been subjected to any stimulation, the Feulgen reaction positive substance was situated within the nucleus. In regard to the behavior of nuclear organelles in this reaction, it was always found that the nucleolus was situated in its central part and the positively reacting structure appeared to be compact. Only a fine network of particles fringed the nucleolus associated chromatin.

As the metabolic disturbance developed progressively, the distribution of ribodesose nucleotides within the nuclei of the hair cells showed a marked difference. The hair cells, particularly outer hair cells, are characterized by a large amount of nucleolus associated chromatin. This stage of the disturbance in the nucleus of hair cells could be found even in its earliest stage, as shown in moderate quinine intoxication which was rapidly reversible as demonstrated by recovery of cochlear microphonics.

Immediately after moderate overstimulation for 20 to 30 minutes, the nuclei were characterized by pyknosis. The detail in this stage was a small nucleus, rich in Feulgen reaction positive substance which was frequently observed in outer hair cells of dihydrostreptomycin administered guinea pigs or overstimulated guinea pigs. As these animals showed fairly complete recovery of cochlear microphonics in most cases, it seems that the cytochemical change found in the hair cells may be reconstructed in a certain amount of time elapse, though in a varying degree.

Dihydrostreptomycin in a larger dosage employed in a series of guinea pigs caused a loss of sensitivity for cochlear microphonics in the audible frequency range, especially in high tones. In these cases

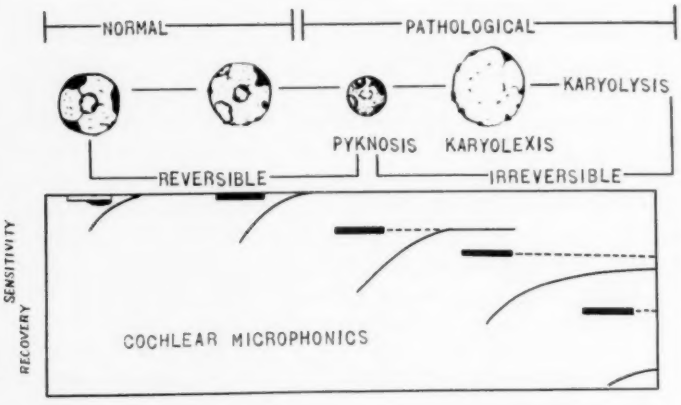

Fig. 12.—Schematic diagram showing correlation between the Feulgen reaction and the cochlear microphonics in various stages of metabolic disturbance in the hair cell of the organ of Corti.

the cytochemical changes found in the organ of Corti were different from the type found in quinine intoxicated or overstimulated animals. The typical changes were found mainly in hair cells of the basal turn. The change which was present in the hair cells ranged from karyorrhexis to complete karyolysis; the Feulgen reaction positive substance in hair cells, particularly in the outer hair cells, was reduced in amount and scattered around the border of the nuclear membrane.

The most marked change was the absorption of cell nuclei and the absence of Feulgen reaction positive substance in outer hair cells. These severe changes were scarcely to be found in inner hair cells. The irregular distribution of the cell nuclei was remarkable, most parts of which showed pyknosis; overstimulation lasting three minutes produced a reduction in amplitude of cochlear microphonics and this deprivation was generally irreversible and often progressed. This probably represented the hair cell injury. It is possible to produce by this overstimulation a permanent damage of hair cells.

In view of both cytochemical changes in hair cells and the recovery process of cochlear microphonics after overstimulation in various stages of intoxication, the course of the change can be graphically shown (Fig. 12). The first change observed in slightly intoxicated

animals occurred in the cell nuclei where Feulgen reaction positive substance was located around the border of the nuclear membrane. In this stage the sensitivity loss for cochlear microphonics cannot be observed and the recovery process in connection with overstimulation was as rapid as in healthy animals. This indicated that these changes are reversible.

In the development of intoxication a large amount of Feulgen reaction positive substance appeared in small sized nuclei. However, this stage as observed in overstimulation was reversible, because cochlear microphonics showed signs of complete recovery. The behavior of the recovery time indicated that there was certainly a decrease in speed of physico-chemical reconstruction in the hair cells.

At the end of intoxication merely a scant amount of DNA (or complete absence of it) was observed in cell nuclei. Reduction in amplitude of cochlear microphonics was generaly irreversible and often progressed with time. This indicated that the physico-chemical disturbance was developed to a point of permanent damage by overstimulation.

SUMMARY

For the purpose of revealing the physico-chemical process in the hair cells of the organ of Corti in guinea pigs, the hair cells from guinea pigs were studied with cytochemical analysis and cochlear microphonics, 1) in acoustically quiet conditions, 2) after acoustic stimulation and, 3) in various stages with quinine or dihydrostreptomycin sulfate.

The hair cells in the organ of Corti in normal cases have a well-developed nuclear system for the formation of protein, consisting of one nucleolus rich DNA framed by an exterior network of small particles, and distribute regularly. Cochlear microphonics shows signs of recovery after overstimulation with 4000 cps 90 db lasting three minutes.

The early stage of intoxication disclosed in the hair cell nuclei is an increase of nucleolus associated chromatin and pyknosis. With moderate stimulation the recovery time is prolonged and the behavior of the recovery time indicates that this moderate stimulation produces a definite cumulative decrease of the speed of physico-chemical reconstruction in hair cells. However, a complete recovery for cochlear microphonics with the repetition of a click is always found. This

shows that these cytochemical changes found in the hair cell nuclei of the organ of Corti are generally reversible.

In the late stage of intoxication with dihydrostreptomycin the cytochemical changes present in the hair cells range from karyorrhexis to complete karyolysis and the arrangement of outer hair cell nuclei is irregular. Moderate stimulation can easily cause the physico-chemical disturbance to become permanent injury. The recovery after over-stimulation is generally incomplete and occasionally the decrease in amplitude of cochlear microphonics progresses, probably due to cumulative metabolic disturbances in the hair cells.

KYOTO PREFECTURAL COLLEGE

This work was aided in part by a grant from the National Research Council of Japan. We wish to acknowledge our indebtedness to Doctors Schida and Shimizu for their valuable assistance in this work.

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VII

VARIABILITY IN THE PLANE OF THE FENESTRA VESTIBULI

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The problem of the stapes[1] and the stapedial locus[2] has been analyzed by this writer. After calculating the related planes of semicircular canals in dissections upon the paired temporals in situ, and with reference to cranial concomitant measurements, it appears that in narrow crania the fenestra vestibuli faces laterally, and in wide crania faces rather more rostrally. This conditions the locus of the stapes very significantly.

In narrow crania the lateral canal may be depressed 10°, or slightly more, distolaterally.

In wide crania the lateral canal may be depressed as much as 40°. The superior canal varies coincidentally; and in narrow crania the two superior canals are at nearly 45° to the median sagittal plane. But in wide crania the two superior canals tend to approach the median sagittal plane, and may correspond to it at 32°. This rotates the fenestra vestibuli (oval window) correspondingly.

The action of the base of the stapes is somewhat like that of a plate, hinged posterolaterally; the rostromedial edge has greater movability, controlled by the stapedius muscle. In embryonic life the middle ear is filled with a thin jelly, which probably prevents much movement. After birth this jelly seems to disappear, but the pull of the stapedius muscle tends to bulge the posterior crus of the stapes. This greater curvature of the posterior crus is noted, but is not always present. The fenestra vestibuli, also, is not only rotated as to its plane, but its rostromedial end is tilted in wide crania, so that the axis transversely is lifted medially, and depressed laterally. This again must be regarded in any effort to mobilize the stapes.

With the tilting of the plane of the fenestra vestibuli, the cochlea also is tilted, so that the promontory is more or less inclined laterally

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at the top, and the fenestra rotunda is rotated and inclined laterally and downwards.[3]

Careful dissections must be made upon a skull base with both temporal bones in situ, to compare angles and planes. The semicircular canals are laid open from within, but not isolated, and the tegmen tympani is carefully removed to allow full inspection; also the tegmen antri. At once the variable relations of the facial canal appear, passing between the oval window and the end of the superior and lateral canals. The edge of the oval window derived from the pro-otic element is more curved than the lower edge which is derived from the opisthotic.

Special attention may be directed to the recent studies of Bellucci.[4] His data are most interesting, with 63 cases mobilized, and 35 probably fractured.

Other writers have been cited in various publications recently; but no illustrations give the variable relations of the stapes in plane, or of the tilting of the oval window.[5]

This brief note is intended to suggest more cranial dissection. This writer has published photostats of enlarged ear ossicles.[1] It is remarkable that from the Lake Pelican area of Minnesota there is a stapes (removed from temporal element) showing two short and perfectly straight stapedial crura. From an Australian cranium of the collection of Dr. William King Gregory, this writer removed a similar stapes. It is not seen in this form in Africans of modern type, nor in specimens of Egyptian provenience of 4000 years ago.[1]

All crania should have the ear-holes immediately closed with soft cotton plugs, until the ossicles are removed, otherwise they will fall out in adjusting the skull.

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VIII

THE PROTEINS OF THE PERILYMPH

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and

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Even today our knowledge of the chemical composition of the labyrinthine liquids is extremely limited. The few data reported in the literature about the physical, chemical and immunological properties of the perilymph and the endolymph appear to be not only isolated, but also difficult to compare owing to the different species of animals used in research and the different methods of analysis employed by the authors.

The main difficulty in the study of the chemical constituents of the perilymph is the impossibility of obtaining sufficient material for analysis from the animals commonly used in laboratories for this research.

In another paper[1] one of us reported a method of taking the perilymphatic fluid from a horse, by which it was possible to obtain comparatively large amounts of this liquid without difficulty. Using this technique, in previous work[2,3] we were able to identify the free aminoacids and the ketoacids of the perilymph. It appeared to us to be profitable to follow this research with a study, both quantitative and qualitative, of the proteins of this biological fluid. Research has been carried out on the protein content of the perilymphatic liquid of cats,[4] of human beings with Ménière's syndrome,[5] of guinea pigs[6] and of sharks.[7] The majority of authors agree that the protein content in the perilymph is less than in the blood serum but more than in the endolymph and in the cerebrospinal fluid. With regard to the different protein components of the perilymph described by Vilstrup and his colleagues,[8] research on sharks and research done by two of us[9] on cats may be recalled. However, the results of these experiments

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are not very satisfactory, both because of the technique used and the animal species chosen.

As we had a method which enabled us to obtain comparatively large quantities of perilymph, we felt that it would be interesting to make a detailed and complete study of the proteins of this biological liquid, carrying out parallel observations on the blood serum and on the cerebrospinal fluid. This was done with the particular hope that research planned in this direction might throw some light on the origin of the perilymph from the blood or from the cerebrospinal fluid.

EXPERIMENTS

Perilymph. Using the technique which has previously been described,[1] the perilymphatic liquid was obtained from horses and mares between two months and three years old, within 15 to 30 minutes after death.

After having removed the heat of the animal, the base of the skull is freed of all the soft parts down to the bone in the region of the petrous bone. Here there is a bony prominence coinciding with the tympanic cavity. When the bony wall which constitutes the lower wall of the middle ear has been removed with small scissors, the formation of this is immediately visible, particularly the round window.

The perilymphatic liquid is removed through the membrane of the round window by means of suction with a sufficiently fine Pasteur pipette with 3 to 5 mm of the end at right angles to the main axis. About 0.015 to 0.025 ml perilymph can be obtained from each ear. The amount required for the individual analyses was obtained by combining the specimens taken from several animals.

Cerebrospinal fluid. The cerebrospinal fluid was taken from the cervical swelling. Known amounts of liquid, taken from the same animals, were combined.

Serum. The serum was obtained from blood taken from the large vessels of the neck. Known amounts of serum, taken from the same animals, were combined.

QUANTITATIVE ANALYSIS OF THE TOTAL PROTEINS

A micromethod[10] based on the biuret reaction and subsequent spectrophotometric reading at 330 μ was used in the quantitative

analysis of the total proteins. The analyses were carried out on 0.2 to 0.5 ml of perilymphatic fluid, 0.5 ml of cerebrospinal fluid and 0.5 ml of blood serum diluted 1:25 with 1 per cent solution of NaCl.

We proceeded as follows: In a centrifugal test tube 4 ml of 10 per cent trichloroacetic acid were added to the quantities of the biological liquids mentioned above. The mixture was centrifuged after 10 minutes. The supernatant liquid was removed and the precipitate was dissolved in 4 ml of 3 per cent NaOH, then 0.2 ml of Benedict's reagent was added. After 15 minutes a spectrophotometric reading was taken at 330 μ against a blank.

In each experiment the analyses were carried out as said before on combined specimens of the individual biologic liquids under examination. Table I gives the values found in the quantitative analyses

TABLE I

TOTAL PROTEINS IN MG PER HUNDRED ML

EXPERIMENT NO.	SERUM	CEREBROSPINAL FLUID	PERILYMPHATIC L.
I	6600	115	326
II	7080	106	281
III	6950	132	270
IV	7320	120	290
Average	6987.5	118.2	291.7

of the total proteins in the blood serum, the perilymph and the cerebrospinal fluid.

The average protein content of the perilymph is equal to 291.7 mg per hundred ml, while the average cerebrospinal fluid is 118.2 mg per hundred ml and for the blood serum 6987.5 mg per hundred ml.

ANALYSIS OF THE DIFFERENT PROTEIN COMPONENTS

This was carried out by electrophoretic separation on paper.[2] Since the perilymphatic liquid and the cerebrospinal fluid have a low protein content, it was necessary to concentrate these biological liquids until a protein concentration of 2 to 5 per cent was obtained. This was accomplished by two different processes, both proving suitable for the purpose. The first consisted in lyophilizing the liquids and then dialysing against distilled water to remove part of the salts; the

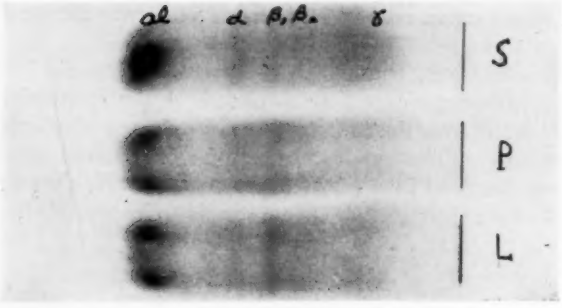

Fig. 1.—Paper electrophoresis of serum (S), perilymph (P) and cerebrospinal fluid (L) of the horse. Veronal-veronal sodium buffer, pH:8.6; ionic strength 0.05; paper Wathman No. 1; dye:amido Schwartz 10 B; time: 5 h.; 300 volt/23 cm.

other consisted of the dialysis of the liquids themselves against "polyvinyl-pyrrolidone" at 20 per cent.

Figure 1 shows electrophoresis of serum, cerebrospinal fluid and perilymphatic liquid of a horse, carried out in parallel on the same strip.

In the perilymph, protein fractions can be recognized corresponding to albumin and globulins α 1, α 2, β and γ. These fractions can also be in the cerebrospinal fluid.

Table II gives the per cent values of the protein fractions of the blood serum, perilymphatic liquid and cerebrospinal fluid of a horse, determined by densitometric analysis. Several electrophoretic analyses were made of each sample of biological liquid so that the single values shown in the table represent an average of at least two analyses.

CONCLUSIONS

Research showed that the perilymphatic liquid of the horse has a protein content of around 290 mg per hundred ml. The analysis of the data from the experiments carried out on the perilymphatic liquid, cerebrospinal fluid and the blood serum enables us to establish that the perilymph has a low protein content compared with the serum, but higher than the cerebrospinal liquid. With regard to the protein fractions, two facts shown by the experiments should be

TABLE II

PROTEIN FRACTIONS IN PERCENTAGES OF TOTAL PROTEINS

MATERIAL	EXPERIMENT NO.	ALBUMIN	GLOBULIN α 1	α 2	β	γ
Serum	I	45	3.5	10	10.4	23.1
	II	50.2	3.3	8.8	15	22.1
	III	55	2.5	9.5	14	19
	IV	52	3.6	10.5	14.2	21
	Average	50.52	3.22	9.7	12.4	21.3
Perilymphatic liquid	I	50.3	2.3	8	26.7	12.5
	II	49.7	4.1	10	26.2	10
	III	50.6	3	8.5	21.4	10.5
	IV	53.5	2.9	11.1	23	8.5
	Average	51.05	3.04	9.4	24.3	10.3
Cerebrospinal fluid	I	48.1	3	7.1	24.9	16.3
	II	51.3	2.75	6.5	23.5	16
	III	49.3	4.2	9.5	24.5	12.5
	IV	47.5	2.7	9	25.5	16
	Average	49.05	3.16	8.02	24.6	14.2

emphasized. The first is the presence in the perilymph of all the fractions found both in the blood serum and in the cerebrospinal fluid; the second is the per cent proportion of the different fractions in the constitution of the protein content of the perilymphatic liquid; actually, in comparison with the blood serum, the perilymph shows a high β-globulin content and a low γ-globulin one.

In previous research[2,3] we have shown that the free amino-acids and the keto-acids are present in the perilymph of horses in quantities appreciably greater than in the cerebrospinal fluid and in a similar quantity to that found in the blood.

The results of the previous and present research both preclude that the perilymphatic liquid could be considered as a simple derivate of the cerebrospinal fluid; but they rather suggest origin from the blood, probably through a selective filtering process.

SUMMARY

The authors have studied, both from the quantitative and the qualitative point of view, the proteins of the perilymphatic liquid,

the blood serum and the cerebrospinal fluid of the horse. The perilymph has a protein content of around 290 mg per hundred ml, an amount appreciably greater than that of the cerebrospinal fluid and much less than that of the blood serum.

Paper electrophoresis of the protein constituents has established that all the fractions existing in the blood serum and in the cerebrospinal fluid are present in the perilymphatic liquid and that, compared with the blood serum, the perilymph contains a greater quantity of β-globulin and a lesser quantity of γ-globulin.

(Received for publication May 21, 1956)

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IX

BILATERAL CONGENITAL CHOANAL ATRESIA: A REPORT OF THREE CASES CORRECTED SURGICALLY IN THE NEWBORN

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Although numerous papers have been written concerning choanal atresia, including a variety of operations for its correction,[1,4-6] there is one feature of the subject which has been receiving full attention in medical literature only very recently, namely, the prompt correction of bilateral atresia in the newborn. Over the years there has been little emphasis upon the important fact that it is extremely difficult for some of these infants to survive the neonatal period, and one wonders how many cases of bilateral atresia corrected in childhood could have been matched by other cases dying shortly after birth of unexplained asphyxia. Beinfield[4] states that as early as 1881, Ronaldson issued the warning that this condition might account for the unexplained suffocation of some newborn infants, basing this opinion on autopsy findings. Nevertheless, it is most rare to find any report of the correction of bilateral choanal atresia in the newborn. Recently, a few have appeared. Hanckel,[5] in 1949, reported a case operated on the forty-first day of life, having already had a tracheotomy on the thirtieth day. The case reported by Beinfield[2] in 1954, however, is more directly related to the subject in point, where prompt correction of the bilateral atresia was the method of relieving the dyspnea. In his case the operation was done on the second day of life by an intranasal approach. Hanckel pointed out that Schweckendiek had reported, in 1937, transpalatal operations on two infants ten days and nineteen days of age. Hough[7] followed in 1955 with the report of a case operated upon five days after birth, also intranasally. His paper includes an illuminating description of the mechanism of asphyxia, demonstrating by x-ray films a sucking up of the tongue and floor of the mouth against the palate on inspiration and a ballooning effect on expiration, whereby the soft palate is forced forward against the tongue.

From the Division of Otolaryngology, College of Medicine, University of Vermont. Read before the New England Otolaryngological Society, Boston, Massachusetts, February 8, 1956.

Three additional cases, presently under our care, are here presented.

REPORT OF CASES

CASE 1. C.N., a baby boy, was born on March 12, 1955, in the Mary Fletcher Hospital. At the time of birth there was slight cyanosis. A half hour later the cyanosis was quite marked but it was promptly relieved by the insertion of an oral airway such as that used by the anesthetists. The baby tolerated this airway and was kept in oxygen with it in place. Several times in the next 24 hours attempts were made to remove the airway but each time there ensued immediate retraction and cyanosis. It was impossible for the infant to take food because of the dyspnea and it was necessary to feed it by gavage. Examination on the second day of life revealed an obstruction preventing the passage of a small catheter through either nasal cavity. The instillation of lipiodol into the nasal cavities established the diagnosis of bilateral choanal atresia.

Forty-one hours after birth, Dr. William Montgomery did a transpalatal correction of the choanal atresia. Polyethelene tubes were inserted from the anterior nares to the nasopharynx and sutured to the columella. There was immediate relief of dyspnea. The infant did very well, though it needed constant attention and frequent suctioning of the tubes, especially after feedings. The Breck feeder was used with good success. The baby was kept in a moist atmosphere and was given penicillin and streptomycin.

During the next two weeks the polyethelene tubes in the nose became obstructed a few times despite constant nursing and it became quite clear that he could not tolerate being without them. On two occasions the tubes came out, once when the suture broke and another time when the suture worked through the skin of the columella. On this second occasion, when the tubes were replaced, a method of suturing them to a tracheotomy tape was developed. The tape was passed across the upper lip and fastened to the cheeks with adhesive tape.

On April 9 the baby was discharged from the hospital, 27 days old, taking formula and cereal well. He was readmitted six days later, the tubes having remained in during the period at home. An attempt was made to remove the tubes but after 12 hours the baby became dyspneic and it became necessary to reinsert them. The infant was discharged the following day. By the time he was six weeks old he was able to go without the tubes for several hours a day, but at least one tube was inserted during the night and also during feedings.

On April 29, the first dilatation of the choanae was done with esophageal bougies up to No. 16 French. Until May 30 such dilatations were done once a week. After each treatment it was necessary to keep at least one tube in for 48 hours. From June 1 to December 1 the dilatations were done on a two-weekly basis and since December 1, until the time of writing they have been done every three weeks.

The baby is now eleven months old and is entirely healthy in all respects except that he still has to wear a tube at night and is having his routine dilatations.

Throughout the months the baby has never been left alone. Its crib is less than 12 inches from the parents' bed. An electric suction pump is kept available and has proven most valuable for prompt relief of nasal obstruction by heavy mucus. The parents are able to remove or insert the tubes as desired. Neosynephrine 0.125 per cent nose drops have been found helpful on many occasions.

CASE 2. J.B., a male infant, was born at term in the Heaton Hospital in Montpelier. It was noticed that the head was misshapen, having the form of a scaphocephaly. The infant cried at birth and seemed to behave normally for two days, but then he developed dyspnea and cyanosis and the nurses first noted that he was breath-

ing entirely through the mouth. It was necessary to keep the infant in an isolette in oxygen thereafter and he continued to have frequent bouts of cyanosis throughout the next two weeks. He took the bottle for the first two days of life but then was unable to feed because of nasal obstruction. He had to be fed throughout this period by gavage.

At two weeks of age he was transferred to the Mary Fletcher Hospital and was noted on admission to be thin and pale. The frontal bone was bulging. The posterior fontanel was closed and hard. The anterior was open widely and bulging. The saggital suture was closed with a ridge of bone. The lambdoid suture was also closed but not smooth. The other sutures were widely opened. The baby weighed only six pounds. He was feeding poorly, was breathing through the mouth and that with difficulty. There was retraction of the upper sternal area. Two days after transfer the breathing difficulty had become so severe that it was of emergency category. Otolaryngological examination at this time revealed obstruction of the posterior portion of each nasal cavity, not permitting the passage of even a small catheter. Lipiodol was instilled into the nasal cavities and it was observed that a tiny trickle of it managed to get through into the nasopharynx. A diagnosis of incomplete choanal atresia was made.

With the use of endotracheal anesthesia further probing of the nasal cavities was done and a tiny opening permitting the passage of a filiform ureteral bougie was found in each side. Dilatations were then done with graduated urethral sounds up to No. 12 French. Polyethelene tubes were inserted and were sutured to the septum. The breathing was immediately relieved. The infant began to eat well and gained weight.

Nine days postoperatively the suture in the septum worked out and tubes were maintained by being sewed to a strip of tape, as in Case 1.

On October 25 a cranioplasty was done. On November 9 the infant managed to pull out the nasal tubes, 27 days after they had been placed. He breathed so well that they were allowed to remain out and he was kept on neosynephrine nose drops and frequent suctioning of the nostrils.

On November 15 a second cranioplasty was done. (Both operations were performed by Dr. R. M. P. Donaghy.)

On November 23, 41 days after the choanal operation, the first dilatation treatment was done. It was carried up to No. 16 French. Urethral sounds were used. The infant was discharged on December 4, three months and one week of age, weighing nine pounds, three ounces. He was maintained at home on nose drops and suctioning with a bulb syringe. Twelve days later he was readmitted and a dilatation treatment was done on the same day. Two days after the treatment there was difficulty in breathing and eating and it was necessary to replace the nasal tubes. These were kept in for three days. Dilatation treatments were then done at two-weekly intervals and after each the tubes were replaced for two or three days. The infant is now breathing well, eating well, and gaining weight and is being cared for at home. The interval between dilatations is now three to four weeks.

CASE 3. S.P., a girl, was born at term at the Barre City Hospital, on November 29, 1955, apparently well-formed in all respects except that it was noted right at the time of birth that the nasal passages were not open. The pediatrician observed dyspnea and cyanosis and attempted to pass a rubber catheter. The mouth had to be held open in order to prevent cyanosis. About two hours after birth Dr. Emilio Gianarelli undertook to correct the choanal atresia. He inserted a small periosteal elevator up to the solid bony wall. He found that on gentle pressure the wall began to give way and he was able to force the elevator through. He then turned this instrument so as to break the bone in other directions and was able to pass through some polyethelene tubes. Attempts to remove the tubes were made off and on during

the next two weeks but it was necessary each time to replace them promptly. At the end of two weeks the infant was transferred to the Mary Fletcher Hospital because of failure to gain weight and continuing dyspnea despite the intranasal tubes. On admission it was noted that the tubes were considerably smaller than those we had used in the previous cases. Consequently we did an immediate dilatation of the choanae up to a size No. 14 French, using the urethral sounds, and inserted larger polyethelene tubes. Two weeks later an attempt was made to remove the tubes but the infant immediately developed new difficulty in eating and they were replaced. Additional dilatations have been done at three and four week intervals.

The infant is being kept at home with the tubes out most of the time, except that she is kept in the hospital three or four days after each dilatation. The parents have borrowed a suction pump and a neighboring pediatrician is available to replace the breathing tubes if difficulty in breathing should develop.

SUMMARY

Following the reports of a few recent authors,[2,3,5,7] we are here presenting a situation which up to the present has been exceptionally rare in medical literature; namely, the correction of bilateral choanal bony atresia as an emergency procedure in the newborn. It is hoped that a greater consciousness of this possibility can be built up in the minds of those physicians dealing with the problem of neonatal asphyxia. The diagnosis is quite simple and the emergency corrective measures are not difficult. It still remains to be seen what later corrective procedures may become necessary before a finally satisfactory airway is permanently established. Thus far, we have been carrying these cases simply on periodic dilatations of the choanae and a rather meticulous program of home care as to feeding, nasal cleansing, and the intermittent use of polyethelene breathing tubes. As stated by Hough,[7] we must stand prepared to correct these cases again at a later date by a more thorough type of transpalatal or intranasal approach. Whether this will be necessary only time can answer.

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X

LARYNGEAL METASTASES FROM MALIGNANT TUMORS IN DISTANT ORGANS

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AND

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Although metastatic disease within the larynx is apparently very rare, the possibility does enter into the laryngologist's problems of differential diagnosis and may increase in importance as more specific information is gained about the behavior and mode of transport of metastatic cells.

In reviewing the world literature from 1916 through 1951, Loughead and Bushnell[1] found four instances of cancer metastatic to the larynx. These authors reported an additional patient with this condition. Three of the five tumors reported were malignant melanomas, probably bearing pigment, and the remaining two originated in renal cells. Lymphomas were excluded from this review. (Multiple myeloma has been recorded four times, plasma cell myeloma numerous times, and leukemic infiltrations are not rare.) To this series may be added the case of a bilaterally symmetrical subglottic metastasis from a renal sarcoma reported by Rebattu, Martin, and Takizawa[2] in 1950, and two cases of metastatic hypernephroma reported by Maxwell[3] in 1942.

Nowhere in the world literature of the past 40 years has there been found a case of carcinoma of the larynx metastatic from the prostate gland. In the memories of staff members in the Departments of Urology[4] and Pathology[5] at the University of Michigan, no such case had ever been encountered at the University of Michigan Hospital before September, 1956. Therefore it was a striking coincidence when two cases of laryngeal adenocarcinoma metastatic from primary cancers in the prostate gland appeared at this Hospital within a period of four months.

REPORT OF CASES

The first patient, an eighty-three year old man, was admitted to University Hospital on September 9, 1956, complaining of pain in the left side of the throat which radiated to the left ear, of several months' duration. His weight had remained stable for some time. The patient had had hypertension for the preceding ten years and a questionable "heart attack" in 1946. The urinary stream had decreased somewhat in size during the months prior to admission and the change was accompanied by a certain amount of dribbling and hesitancy, and increase in frequency of urination. There was no history of musculoskeletal pain.

Physical examination showed a blood pressure of 200/95. There was a pedunculated, ulcerated, superficial red mass, 1 cm in diameter, arising on the upper surface of the left arytenoid eminence. There was no apparent abnormality of the vocal cords. No cervical nodes were palpable. There was a right inguinal hernia, controlled by a truss. The prostate showed enlargement and induration considered to be typical of carcinoma. Laboratory examinations included a hemoglobin level of 15 gm per cent and a white blood cell count of 7,150. Results of urinalysis were within normal limits. The acid phosphatase level was 2 King Armstrong units and the alkaline phosphatase 3.7 King Armstrong units. The electrocardiogram was considered to be within the normal range. X-rays of the lumbosacral spine and pelvis showed only hypertrophic spondylitis and arthritis of the hips. The chest x-ray was interpreted as normal. On September 18, 1956, bilateral orchiectomy and punch biopsy of the prostate were performed. The prostatic tissue submitted to the pathology laboratory was diagnosed as "small atypical glands, giving rise to a suggestive diagnosis of adenocarcinoma." On September 13, 1956, the laryngeal lesion was biopsied by indirect laryngoscopy. This tissue consisted of a papilliferous adenocarcinoma growing beneath stratified squamous mucosal surface. The patient was discharged on stilbesterol therapy, 5 mgs daily, and almost immediately noted a reduction in the urinary obstructive symptoms.

The second patient, a seventy-four year old man, was admitted to University Hospital on January 2, 1957, with a questionable diagnosis of carcinoma of the thyroid gland. The patient gave a history of a mass in the midanterior cervical region for the past one and one-half years which had increased in size during the four or five months before examination. In addition he mentioned gradually increasing hoarseness over a period of approximately twelve months. His past

history was non-contributory; specifically, the patient recalled no symptoms that would seem to involve the prostate gland. Examination of the ears, nose, and throat showed no abnormalities except a nodular hard moveable mass, 3 x 4 x 6 cm, overlying and adherent to the thyroid cartilage on both sides. The interior of the larynx was normal except for slight enlargement of the anterior third of the left true vocal cord and ventricular band. The cords moved, but motion of the membranous portion of the left true vocal fold was slightly restricted. There were no palpable cervical nodes. The thyroid isthmus could be felt below the mass. The remainder of the physical examination disclosed a blood pressure of 170/70, bilateral immature cataracts, a large direct right inguinal hernia, and a firm, nodular, somewhat enlarged prostate gland which the urological staff members considered definitely indicative of carcinomatous change. There were severe varicosities on the left leg. Laboratory studies showed a hemoglobin level of 13.2 per cent and a white blood cell count of 7,500. Urinalysis was normal. Phenolsulfonphthalein retention was 18 per cent in fifteen minutes. The acid phosphatase level was 23 King Armstrong units and the alkaline phosphatase level 3 King Armstrong units. Bromsulphalein showed 11.6 per cent retention in 45 minutes. The two hour postprandial blood sugar level was 101 mg per cent and the nonprotein nitrogen level was 41 mg per cent.

Radiographic examination of the chest, spine, pelvis, and soft tissues of the neck were normal with the exception of hypertrophic arthritis of the cervical and dorsal spine and slight irregularity of the left laryngeal ventricle.

On January 21, 1957, a biopsy of the mass involving the thyroid cartilage was performed under local anesthesia. Pathological diagnosis of this specimen was "moderately well-differentiated adenocarcinoma." On the same day, bilateral orchiectomy was performed. After an uneventful postoperative course the patient was discharged on a regimen of 5 mg stilbesterol daily.

COMMENT

With these cases added to the previous total, there have been reported ten cases of malignant tumors arising elsewhere and metastasizing to the larynx. This seems to be an incidence considerably smaller than one might expect on a random basis. Does the larynx therefore have an inherent resistance to the propagation of the seeds of distant tumors? Is the remoteness of this organ such that viable blood- or lymph-borne tumor emboli are unlikely to lodge here?

Accepting the two cases reported above as bonafide single metastasis from carcinomas of the prostate, what then was the route by which the tumor emboli reached the larynx?

It has been demonstrated that neoplastic emboli will lodge and grow in any organ where they accumulate in sufficient numbers.[6] The proclivity of the larynx for development of epithelioms is well known. There is nothing in the histological character of this organ to suggest an impediment to the growth of virulent tumor cells.

The systemic vascular network supplies the larynx generously with blood filtered in the capillary beds of the lungs and liver. It is quite probable that these two organs retain a great deal of particulate blood-borne substances, specifically tumor emboli.[7] An alternate route by which the cancer cells might have traveled from the prostate to the larynx is the vertebral venous system. Batson has described the possible role of retrograde flow in these channels with relation to the hematogenous spread of malignant growth without passing through the lungs.[8] Finally, the lymphatic connections of the larynx are such that advent of lymph-borne neoplastic emboli from distant sites is extremely improbable.

SUMMARY

Medical literature since 1916 records only eight cases of tumors metastatic to the larynx from distant organs. At the University of Michigan Hospital two cases of carcinoma of the prostate gland with solitary laryngeal metastases have been encountered recently, bringing the total to ten. Despite the rarity of metastatic disease within the larynx, the laryngologist does well to heed the biopsy specimen which appears to be at variance with the general picture of tumors arising primarily in the larynx.

University Hospital

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XI

RES MEDICA, 1956

THE WHERRY LECTURE*

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ST. LOUIS, MO.

In the midst of this busier week of our busy lives, it is fitting that we pause now, for a brief hour, to give thought to an old friend who left us some fourteen years ago. Having known Bill Wherry intimately during the last two decades of his life I am grateful to the Academy in a very personal way for the invitation to deliver this lecture and to do him honor.

Those of you who worked with Bill remember him as a compelling character: studious, thoughtful and alert, and dedicated with all his heart and soul to the progress of the Academy and the American Board. His interest in the details of medical practice was overshadowed by a consuming occupation with its trends and tendencies: with teaching, examining and improving standards. He burned many a midnight kilowatt over his beloved charts and tables and statistics.

Were his spirit of a mind to descend from its bright cloud today and spend an hour here with us, there is no doubt that he would clamor first after these things—the Res Medica of 1956. So it seems appropriate to me to desert the laboratory and the sickroom this year, and to discuss with you and Bill the special characteristics of contemporary medicine, which engrossed him, as I see them today.

You will soon discover that this will by no means develop into another "recent advances" report. Medicine today, as in other ages, is not all advance; nor is it all regression. It is a confounding jigsaw puzzle of progress, stasis and decay; of inspiration, faith, cynicism, trial and error, with a liberal seasoning of socialism, politics, stock

* Read before the Academy of Ophthalmology and Otolaryngology October 17, 1956.

markets, news releases and an amorphous hodge-podge of heroism, sacrifice, self-aggrandizement, spiritual hurricanes and miracles.

I shall not presume to pass judgment, but shall attempt only to fit the pieces together as accurately and realistically as they will permit, leaving for you the fun of pondering the completed picture.

* * *

Never since the world began has Medicine been so much the pocket-piece of the young mother, the cancerphobe, the politician, the professional promoter and the stock-broker as it is today. Unless the medical man promptly increases his stature, and that in a big way, he will soon become merely the maypole around which the laity is gaily dancing, and will inevitably end up by finding himself completely fettered in gaudy ribbons.

Pursuing, then, the Wherry method let us stand off a piece and try to decide just what it is that characterizes this decade. Is it gay? or mauve? or roaring? or atomic? — or what is it? Probably no era was ever able, during its lifetime, to pick its own proper epithet. The best we can do here is to set down the facts and let future generations put a tag on them. We can observe and record, and go into conference.

If one were to stop a hundred doctors here in the lobby and ask them how Medicine got the way she is today most of them would quickly reply, "Sulfonamides, vaccines and antibiotics." How right they would be I leave to you. I doubt that more than a dozen would add these six other cardinal causes:

War

Taxes

The law of supply and demand

Paternalistic trends in government

The high cost of pharmaceutical processes

The daily press.

The six are so closely inter-related as to make the task of separating them almost hopeless. I will try.

* * *

It is apparent that the last twenty years have witnessed a complete change of scene in doctors' offices—what remains of them—and in sickrooms—what remains of *them*.

The doctor-patient relationship has become more tenuous. The doctor's daily schedule has changed. So has his attitude toward prescriptions, laboratory tests, operations, house calls, office procedures, hours, fees and publicity. The disruptions of war lasted so long that many younger men are not even clearly aware of their existence, let alone their magnitude, and the importance of resulting changes, both good and bad.

Let us study our chain of influences link by link, beginning with War. Pearl Harbor caught us half-dressed. Suddenly and frantically everything we possessed was poured into the hopper of hasty preparedness, including our young medical men. For several years—more than a medical school generation—graduates, with scarcely time to read their diplomas, dashed for the nearest transport, hesitating only long enough to get married by the way, and plunged headlong into field medicine.

Field Medicine in War II was incomparably better dressed than her grandmother in War I, but she was still field medicine, with the inevitable emphasis on adaptability and ingenuity, which was all to the good; and on mass tactics, quick turnover and general oversimplification which was not so good, and as training for budding private practitioners was a far cry from the personal tutoring their fathers had enjoyed. When his patient got out of hand the oldster knew pretty much where to look for trouble, because he had known the sick one in the heart of his family and grown up with him medically. The field trained youngster in the same fix met the problem in the only way he knew: he rang for room service in the shape of a psychiatrist or sent the whole bundle down to the base lab. In his formative years, the simple doctor-patient amenities were left out. This made for self-confidence and initiative—and for a good deal of the brashness and fumbling so characteristic of the self-taught.

* * *

By the end of the unpleasantness good years had gone by and something strange had come into the picture: little mouths to feed, big rents to pay, and, with a capital T, Taxes.

Taxes affected him as it did the rest of medicine in two different ways: on the one hand, his own went up and had to be paid; on the other, the excess profits tax and related contrivances all but put an end to private endowments. He finished his graduate training at the expense of you and me, which was completely what he had coming to him, but his mind was not in it. The first G.I. graduate courses after the war were heartbreaking; with one cerebral hemisphere on the recent horrors and the other on feeding a new-found family, much of the teaching settled in the cerebellum as a kind of conditioned reflex. These men will tell you that later on it took years of application and self-discipline to catch up.

* * *

What came to the rescue of the young doctor fresh from service was the next link in our chain, namely, the law of supply and demand. The current short supply of medics kept his stomach from growling but it left a smudge on the complexion of his doctor-patient relationships which only that same law will eventually wash away.

The shortage of medical men, which still exists, permitted returned doctors to establish themselves in practice overnight. In a surge of misplaced sympathy I once approached one of my young friends, only a month out of the medical corps, with an invitation to take over some of my work, but was turned down on the ground that he was too busy with his own practice. It was true; he was!

The experience of "building up a practice" has disappeared. In a ready-made practice, sprung full-panoplied from the brow of—shall we say, Mars? it is difficult for even the doctor himself to find out whether he is any good or not. Patients have become less discriminating, if more demanding, but so have doctors; and since all doctors are prosperous, good ones are less conspicuous.

The war left its mark on the hospitals too. Even topflight institutions were compelled to resort to medical student externes scarce far enough advanced to utilize the teaching material at hand, and certainly incapable of treating the sick. Hemorrhages went undetected, bowels unmoved. Residencies in the best teaching hospitals went begging after the war, while applicants flocked to third-rate places offering good pay. Teachers and teaching facilities which had taken years to build up died on the vine. Some of them are still dead.

* * *

The busy, busy doctor now has two inexhaustible allies which materialized like genii out of a bottle while he was away at the wars. Their names, while not exactly oriental, are high-sounding enough to work magic: Sulfonamides and Antibiotics.

Unquestionably the greatest single advance in medical therapy in the fifteen years since Wherry lived—indeed, in the sixty years since Pasteur lived—is the antibiotic in its various forms. The story of its accidental discovery, the "latent" period before its full significance was realized, the period of waiting while it was reserved for the military and its final emergence as the modern miracle—all this is familiar ground. What concerns us here is not so much the effect of antibiotics on disease germs as on doctors and patients and the traffic between the two.

The birth of penicillin was highly dramatized by its effect on war casualties, an effect greatly heightened by its inaccessibility and high price. The public was aroused, as it has been, impartially, by every medical novelty from Mesmer's needles to ionization. The man in the street quickly decided that lethal infection ("Is it *strep*, Doctor?") was a thing of the past; but right behind him came his doctor, himself flabbergasted by clinical miracles on every side. Among them, the acute mastoid with its emergency operation vanished overnight. The old strategy against sepsis collapsed. A new one arose in its place and doctors, being human (some of them even more human than others), allowed themselves to be inoculated with the uncritical faith of the masses in the miracle. As its cost came down the drug was apt to replace the careful diagnosis. On the whole the human race was doubtless way ahead, but the vigilance of the medical mind relaxed. Was all this years ago?

How about 1956? As in the case of the airplane, the radio and the cyclatron, yesterday's miracle is today's commonplace and the two mightly A's, aspirin and antibiotics, are now side by side on the shelf and, alas, in the mind. The latter still requires a prescription, and a little of the old glamour still clings to it. True, the practitioner, when he finds himself beyond his depth, still refers his patient to the specialist; but how often can he resist giving him a dose of penicillin first, as a parting shot?

* * *

In 1880 sixty-four persons graduated from medical schools per million population. By last year this figure had dropped to forty-two.

In the same period the medical schools of the country had receded from one for each million population to one for each two million. The principal adjustment in this proportion occurred during the first decade of the century with the weeding out of inadequate and unethical teaching institutions and the general clean-up in medical standards.

It is edifying to report that these standards, so stoutly defended by the medical examining boards, and by Bill Wherry who gave much of his life to the task, are still being guarded by the forces which he helped to set in motion.

The effect of governmental and economical influences (the two go hand in hand) upon medical schools requires some mention. Just as there is a shortage of medical men there is also a shortage of medical schools and of money in the schools. There are at present, in this country, 75 four-year medical schools with an enrollment of 28,583 students. Approximately 7000 were graduated in 1955.

Forty-two schools are privately owned, 37 state owned, and three municipally owned. Nearly half (48.7 per cent) of the total operating expense in 1955-6 was met from public funds.

Medical teaching, on the whole, is on a high plane. Graduate teaching is adequate in most places, brilliant in others. "Quickie" courses in this-and-that (mostly that) are ubiquitous. As in other phases of cultural activity towering individuals are becoming fewer and fewer; mass methods are the order of the day. Text books are written by groups instead of Titans.

While somewhat above the level of wages in other university departments, the income of medical teachers is not high. To ameliorate this defect a little the Commonwealth Fund, within the year, announced unrestricted grants of $7,150,000 to ten university medical schools "to meet the urgent needs of strengthening medical education in the nation"—this in addition to $2,000,000 already allocated for the year. The whole picture of professors' salaries was thrown into prominent relief last December by grants to colleges and hospitals across the country totalling a half billion dollars—the gift of the Ford Foundation.

Full-time professors, on the whole, are at a disadvantage, compared to their intellectual equals in business or in private practice. In most institutions there is a working arrangement which permits part-

time private practice, to enable capable teachers to make an adequate living. Without this the run of professors can easily become substandard.

A meagre stipend is apt to attract only a few dedicated persons (with or without private purses) or more often young, untried people whose scale of living still fits into the available salary. The inexperienced youth, trying hard to fill a chair before his feet can reach the floor, may resort to loftiness to fill in for ability for the nonce, and through insecurity avoid seeking help from the elders in his department, only to discover one day that the nonce has passed, the opportunity for greatness is gone, and only loftiness remains.

Nothing is left but an ivory tower into which he climbs until Time and a kindly Emeritus blow him into a genteel sort of limbo, to the great relief of the Department.

I have often pondered the curricula of medical schools and wondered how they get the way they are. I can only explain some of them in terms of my Aunt Ella, long since gone to her reward, leaving behind, by the way, a pair of very instructive otosclerotic temporal bones.

Aunt Ella was a distinguished pianist. She scorned ragtime, which seemed to me a pity. She felt it was too easy; she could make it up, yards of it, any time she felt like it, and would proceed to demonstrate. The result was certainly easy; it was certainly terrible but, primarily, it was certainly not ragtime. With all her musical erudition she just didn't know about ragtime.

It's much the same thing when a curriculum designed to prepare men to practice medicine is concocted in large measure by chemists, biologists and anatomists who not only never practiced medicine themselves but often never even studied it. They just don't know about doctoring.

Much of this argument applies also to the autonomy of special departments in hospitals and schools. Keeping the surgical specialties subservient to the general Department of Surgery and dependent upon the head of that department for time, space, funds and even surgical judgment, dates back half a century to the era when surgery was next to godliness (on the upper side) and the "brilliant young surgeon" enjoyed something akin to the divine right of kings. There

was something to be said for this paragon. There were not many of him in the community; he was well-trained and he knew it. He also knew that most of the specialists in his hospital lacked fundamental surgical training and had few opportunities of getting it. He was horrified—often justifiably so—at their notions of asepsis; he appointed himself to keep a watchful eye on them and it was just as well he did.

Things have changed.

Surgeons are—while not exactly a dime a dozen—let us say, fifty cents, which is in no sense derogatory but denotes abundant supply. Glamour has worn thin. Ability in surgeon and specialist alike is taken for granted. A truly able specialist rarely finds himself subservient to anybody. Mutual respect between surgeons and specialists must make for autonomy. If it fails under this condition, look for greed in some form. If by chance you find a surgeon in one department barring those of another from witnessing his operations, your diagnosis is made.

* * *

Personal publicity and the struggle for a place in the sun fail to call forth the contempt that they used to. Time was when a doctor's picture in the paper (for anything short of his demise) or a personal interview with a reporter called for something speedy and drastic from the local ethics committee. Remember? The change took place when the full-time medico circumvented all this by degrees on the comfortable theory that, since he was not in private practice, airing his little triumphs in the Sunday paper did not somehow apply to him. Right behind him his Institution, at the moment seeking new funds, found the publicity helpful and not only adopted his point of view, but abetted him with its full resources.

Some medical men in 1956, occasionally even a near-first-rater, think nothing of permitting their portraits to grace the covers of some throwaway magazine devoted to transcripts of articles warmed over from the straight-line journals and a pound or so of ads.

It is notable, in this era, also, that the patent medicine is accepted by all doctors; "ethical pharmaceuticals" is the term used now to designate medicines presumably not advertised to the laity. The doctor has always been assumed to familiarize himself with the composition, pharmaceutical and pharmacological characteristics, as well as the incompatibilities and dangers of the medicines he prescribes,

and is not relieved of this responsibility whether it is his own prescription, or a patent pill. The welter of new drugs and their polysyllabic labeling render this nigh impossible. Three years ago there were no less than 140,000 medicaments on the market. 14,000 were added in 1953 alone!

The present tendency toward patent medicines had a very natural beginning. In the days when the physician composed prescriptions to suit himself the elements resided in rows of ornamental jars on druggists' shelves. They were often grown, plucked, pounded, triturated, extracted and filtered by the pharmacist himself.

The manufacture of the complex synthetics of today requires a terrific capital expenditure, processing, standardizing, sterilizing, advertising. When a new type of medication is discovered a rather characteristic symptom-complex sets in among the drug trade. It is often readily diagnosed as, for example, in the case of the vasoconstrictors, the antihistamines and the sulfonamides.

Once the new drug shows evidence of long-term usefulness Boards of Directors everywhere sit up in their chairs and lay plans for a share of the market. Laboratories begin to fiddle with the original formula in search of a related compound which will work as well, let us hope, or even better, or have fewer drawbacks, or failing these, will at least be sufficiently different from the original to be patentable and rate a trade name of its own. Sometimes a mere shifting of a hydrogen atom will do the business.

In the beginning this is all to the good, because the first modifications have a good chance of being improvements, but when the point arrives where there are more manufacturers than modifications, there is standing-room-only and tail-enders appear beyond the pale. Obviously in a group of similar products some are bound to be much better than others, but this will never show up in the publicity and the doctor is hard put to recognize all their names, let alone their qualifications. The specialist, having frequent use for one group of drugs, may ultimately be in a position to judge for himself; the general practitioner, a more occasional user, is almost compelled to rely on the detail man —in person or in print.

In a recent issue of one of the "throw-away" journals no less than 173 proprietary names were advertised. Comment was, of course, all favorable.

The manufacturer, to stay in business, has to market his product so as to insure its reaching the patient in top condition, and competition compels him to make it very attractive. An English journal has recently decried this beautification of adult drugs since apparently it induces children to eat them and poison themselves!

In terms of overhead, the druggist is understandably in favor of the package deal, bottle or pill, for counting tablets from a stock bottle requires fewer man-hours per diem than compounding prescriptions.

As to the patient, he (and especially *she*) loves a new capsule and appears to find special satisfaction in a two-toned perle of great price. A woman recently returned to my office, after having a new prescription filled for a dollar and a half, to caution me that if I was trying to spare her pocket-book, to forget it. She could afford six or eight dollars, like the last time, and was all for quick results!

Is this progress? Just possibly it could be, for it is not at all axiomatic that the doctor of a generation ago who composed his own complicated prescriptions, varying drugs and doses to fit the case, was either very original or very intelligent about it. Most likely the busy practitioner of that day also soon settled back upon a pretty routine list of formulae, either devised by himself or read out of a book, and the patient, then as now, survived.

Still the treatment should have some relation to the disease or be in a degree appropriate to it. A pearl necklace is very well in its place, and a pretty place it is; but for tying up a bundle a piece of string is better.

Salesmanship has created fashions in drugs, as it has in clothes, hats, shoes and motor cars. A decade ago, while we were still bejittered by the ravages of war, and its aftermath, and had nothing to fear but fear, it was the vogue to keep a fine edge on wakefulness, through the use of amphetamine or one of its many relatives, to the point of bootlegging it to truck-drivers on the highways.

Today, when we have less to worry about than at any other time in the past two generations, the accent is on keeping calm, through chemistry if need be. If you have a tilt with the bill collector, or get out of bed on the wrong foot, it's no good taking it out on your wife. You can keep sweet tempered with a small dose of ataractics

on the bedside table: say Thorazine, or Rauwiloid, if you prefer, or Equanil, or Serpasil, or Pacatal, or Frenquel, or Sparine, or Reserpoid, or Raudixin, or Atarax, or a small pinch of Miltown.

Now will you be quiet?

Two Oregon psychiatrists (Dickel and Dixon) made a few astute remarks this year before the clinical assembly of the American Medical Association in Seattle which might very profitably be posted in the country's newspapers, buses and radio programs where he who runs may read. To quote only two: "In the last 25 years we have all seen a philosophy develop that is focused on the need for us to have freedom from several things, including fear. . . . But if we study the natural history of mankind we cannot help noting that tension, alertness, alarmedness, fear, worry, anxiety and apprehension have been, are, and always will be important elements in the shaping of progress. . . . As a nation we Americans have been strong and we envision continued strength because we have been able to rise to the occasion of defending ourselves in the presence of stress tensions and apprehension. . . . We see a malignant tendency invading our thinking, forcing us to believe that no one should ever be afraid, no one should ever feel anxiety, no one should ever feel so moved about his position in life as to do something about it.

"Many of the individuals who are unable to handle the tensions of modern conflicts in philosophy can do so under the use of the tranquilizing drugs. But the majority of these people are doing so without being of real value to themselves and to the group in which they work. [The patients themselves often] felt obvious dissatisfaction with the use of tranquilizing drugs . . . for it seriously impeded their contributions to their company or their value to their group."

And further, "The doctors rather than the drugs should perhaps be considered at fault."

* * *

When I left medical school my respected professor, George Dock, delivered this parting remark: "Good-bye and good luck — and remember, don't learn your graduate medicine from the detail men." Many times have I recalled this warning as I plowed through advertising pages or answered the ring of the pleasantly voluble young man with the satchel and the samples, and I can picture the consternation of my old chief if he could riffle through the advertising pages of the world's foremost medical journal today, and discover tucked away

there another private little medical journal complete with papers, and references, more beautifully illustrated than most others can afford, down to its own little cover, cartoons and advertising, edited and *copyrighted* by—none other than the detail man.

Verily this one has come into his own. That the articles could conceivably be slanted to augment sales, he may stoutly deny, and possibly with some truth. A glance at his balance sheet for last year, however, could easily lead one to the conclusion that this is probably the best paying medical journal in the world.

Medicine is Big Business and one can learn a good deal about it from the financial pages. Wall Street is rubbing shoulders with Harley Street and Park Avenue and for any slight abrasions this may cause there will no doubt be an expensive ointment.

The lead article in a little monthly investment journal, recently, was on a subject now very much in the medical mind, viz., geriatrics. It runs along for quite a number of pages of pure doctor talk, then mentions by name ten new drugs and the fortunate firms who make them. Another financial paper is worried by the fact that poliomyelitis "behaves so erratically—up one year, down the next." The Wall Street Journal cites "medical alarm" as one of the factors in the falling off of the seven-billion-dollar do-it-yourself industry. It seems that a certain medical man reported that every Monday he is sure to have three or four men in for treatment who have fallen off ladders, or cut hands badly, while doing something themselves.

Several months after the initial polio vaccine hullabaloo Barron's Weekly sagely and cautiously observes that "the Salk vaccine appears to be proving out" — noting that the profit margins based on the current price structure were excellent, the druggist buying 10 cc for twenty dollars and selling it for thirty-three.

Triple play: Park to Madison to Wall!

Not long ago a headline in the morning paper read, "Medical Report Sends Stocks Up" and another in an adjoining column, "Polio Fight Sold Like Hucksters Sell Soap." In the evening paper was a four column ad for a popular TV show, called "The Ion Knife," sponsored by a tops drug house together with a tops medical society.

My stockbroker rolls off the names of the new drugs like a medical student and I sometimes think he knows more about them.

In an age when the man—or the boy—in the street has only to switch on his television set to hear (and see) all about the menopause, he learns early the facts of life.

One of our favorite cartoonists draws a young mother saying to her husband, "Don't you think it's time you were telling Junior the story of the bees and the flowers?" and father replies: "I did, and he advised me to consult a psychiatrist."

I do not criticize; I am only reporting to Bill Wherry what I read in the paper in 1956.

* * *

By sheer force of numbers the public, its medical background recently acquired at the news-stand, plays an important part in steering the res medica. It wants only a few random statistics to put itself on record regarding almost anything at all; and many fantastic convictions grow and persist, regardless of the fact that they are based upon demonstrably false extrapolations.

For example: on the slimmest evidence it promptly accepted the dire relationship between tonsillectomy and poliomyelitis, which trained observers, after months of investigation, never established beyond the common sense point of avoiding the operation in the midst of a known epidemic. In this it was encouraged by some rather special pleading from interested quarters.

The drawing room controversies concerning cigarette smoke and its possible carcinogenic properties is another case in point. I have personally not the slightest doubt that excessive smoking may induce lung cancer, but this is based purely upon a loose association between the evidence of such effects of tars on certain tissues, notably skin, and the likelihood that an as yet undetermined amount of tar must be deposited upon the respiratory mucosa by a couple of hundred puffs of cigarette smoke p.r.n. To date I have uncovered no experimental work that offers anything more definite. Incidentally my own observations in the laboratory (not connected with cancer) would suggest that a very infinitesimal part of the tar from inhaled smoke reaches the mucosa and remains there. What I think is immaterial. We are discussing the public and its presumptuous snap judgments.

It is quite ready to accept the fact that two and two make twenty-two, which in a sense they do, if you happen to be a typog-

rapher; but if you are a banker such arithmetic could land you in jail. We are put in mind of an engineering professor's pithy anecdote to drive home the fallacy of extrapolation from insufficient data:

A woman fashionably attired in a long dress (that dates her) got onto a streetcar. She paid a ten-cent fare (and so does that!).

A second woman, in a knee-length garment, paid only seven cents.

A third one wore shorts. She paid only three cents.

The fourth and last one didn't pay a cent. From the data available you might reasonably conclude that she was nude, but there you would be in error. This lady had a transfer.

* * *

Much is being written about medicine by medical men and by as many laymen as can spell the words, and journals flourish. The content and the nature of the writing reflect the prosperous times, and to a certain extent the way that money is now being distributed for research by governments and foundations of great wealth. Almost gone is the lonely, dedicated worker, with stars in his eyes and sausage for his dinner, giving his last ounce of strength and brains for science.

Research has become largely a community affair in which a group of specialists and technicians can cluster comfortably about the great Brain who heads the project—and the papers—and who may be quite unfamiliar with the minutiae of the processes which enter into his Summary and Conclusions.

Until recently multiple authorships were not the rule. In a selected list of journals, in 1930 there were 19 per cent; in 1955 there were over 41 per cent. Like the group tendency in teaching, mentioned above, this group research can easily develop the lack of personal responsibility which is the weak spot in almost anything that is managed by a committee.

Medical literature has always harbored a small percentage of "pot-boilers"—articles with nothing much to say, but written for personal propaganda, or to accommodate some secretary desperate to fill a program, or for any other reason other than the legitimate one of having something new and progressive to impart.

In late years sizeable research grants from governments, foundations and corporations have bred a new kind of pot-boiler. These grants running into four or five figures, entailing employment of several full-time technicians and constituting much, if not all, of the income of the principals, are often on an annual basis and demand RESULTS to insure renewal. In the very nature of things results are often unspectacular, but the show must go on, and it's publish or perish. This accounts for a good deal that is repetitious in the literature and its earmark is the line of fine print at the bottom, which reads, "This work was aided in part by a grant from . . ."

Changes have crept into the semantics, too. New proper names now attach to old proper procedures, whose original authors abode their destined hours and went their way. The old familiar terms now wear new plastic covers: what used to be a mere "effect" is now an "impact." A former "subject," or "aspect," or "standpoint" is now an "area," as "a troublesome *area* in diabetes"—"in the *area* of circulatory disturbances." "Spectrum," a refugee from optics, first attached itself to antibiotics (in the broad sense), then moved on to practically everything else; the and/or fraction of the old insurance blanks and other legal fine print is now high fashion. These are what spicy old Fowler (the English stylist, not our respected colleague) puts down as the "elegant variations of second-rate writers, intent . . . on expressing themselves prettily." Voluminous footnotes have finally been recognized as window-dressing and discarded.

Illustration is blooming with improved engraving processes and improved bankrolls, and above all, with the timely persuasion of specialists trained in medical drawing and photography.

* * *

Cult still follows cult. People who in other ages ran to Mesmer, Fletcher and Coué now bury their feet in mud, or radioactive fall-out, or whatever it is.

Automation has not hit us yet, but it will. I can hardly wait for the automated tonsillectomy which is just around the corner, with electronic circuit-breakers to stop the machinery during the polio seasons. I rather think the gentle human touch will have to linger for a while in the surgery of the sinuses—except maybe the maxillary.

And, by the way, what's become of Dr. Kinsey?

* * *

It has been popular with medical advertisers to include a few, more or less impressive, references to the literature in their copy. With advertisers of high ethical standards these are all that they appear to be; with others they are often the result of what might be called "instigated research." A doctor, usually a young clinician, is asked by the manufacturer to carry out some "clinical research" on his product, and to write a paper about it. A generous batch of medicine, later referred to as having been "kindly supplied by the J and X Company," and a check for a few hundred dollars, not later referred to, are given him. There is nothing remotely reprehensible or unsound in the arrangement except that the young man is seldom chosen for his research training, and in the rosy atmosphere of having been selected to do this important bit of research the results are apt to reveal in the product an astonishing superiority over those feeble and nameless old last year's competitive products, "A," "B" and "C."

If the paper is any good at all, and an editor can be induced to accept it, it can legitimately be quoted in the ads and the publicity well repays the small investment.

* * *

Never was there another such fantastic story in medical annals, nor one which more completely typified the workings of Big Medicine (in the sense that one speaks of Big Business), than the one which broke on the morning of April 12, 1955.

On that day the newspapers carried the word to every house and hamlet that a potent poliomyelitis vaccine had been discovered. The story burst with all the fanfare that Big Advertising could think up, which, to understate it, was quite a burst. Every stop of the organ was pulled out, but in the paean of praise which issued forth there could presently be heard some sour notes. It was a field day for the Press, in an otherwise quiet season, and they made the most of it.

For weeks announcements and retractions, statements and denials, political incriminations, medical dicta — ex- and intra-cathedral — and, of course, the usual profundities of the columnists vied for headlines. Here we had what may be considered a laboratory specimen of a current phenomenon which rates a few paragraphs in this report to Bill Wherry.

It is the phenomenon of a group of laymen engaged in subsidizing an individual disease, and all the corollary medico-social-financial

issues and activities which that implies. In contemporary terms the machinery includes an administrative, fund-raising "Foundation" with a high pressure sales organization, local and national campaigns, astronomical finances, and among other things a lavish distribution of medical grants. Sparked by emotional appeal of the afflicted, and a universal call, not for dollars, but for mere dimes, it reaches for every pocket and every conscience. It is an amazing example of the power of advertising in America. In a recent news release, the Foundation announced "a total of $21,562,456 (of) the March of Dimes funds authorized since 1938 for the National Foundation's comprehensive program of professional education, the largest ever undertaken by a voluntary agency. In all, 5,334 scholarships and fellowships have been awarded by the National Foundation from 1938 through August 30, 1955."

Polio attacks not over 38,000 people annually in the United States and only a fraction of these suffer disabling results. One writer commenting upon this says, "Statistically more than three times as many people in this country die of homicide as die of polio," and adds, "a vaccine is a good thing to have; so is a bullet-proof vest—and the chances of the average person needing the polio vaccine are no greater than of his needing the bullet-proof vest."

In 1954 alone eight such agencies financing separate "fights" against individual diseases raised a total of $140,037,029. The cost of raising these funds ranged from $96 to $140 per thousand dollars —9.6 per cent to 14 per cent.

The extent to which professional publicity and public relations firms now enter into things medical is exemplified by a news release recently received from one of them announcing an event of no more public interest than the coming annual meeting of one of our national special societies. The sales approach which induced such a society to spend good dues on something it needed "like it needs" a trepanation must have been hypnotic indeed.

It is not uncommon to encounter technical medical information in the morning paper at breakfast, which will only pop up in the medical press some hours—or days—later at the office. I recall that when one of the early antihistamines was announced three lady patients asked me for it on the same morning before I had got around to opening the mail to find out what it was. It seems that news of the Great Discovery had leaked out at a bridge party the previous after-

noon, when the hostess had written down for each of them the magic word: TORANTIL.

* * *

As so often happens, we have devoted a very small part of this hour to the sorry wight to whom the whole shebang of medicine is supposed to be devoted: the patient. Where he stands at the moment, except that he actually lives longer than he used to, is not too clear. That he has more and better medicines and diagnostic tests available to him than ever before is incontestable; whether they are selectively and intelligently brought to him on the basis of human sympathy and a deep understanding of his personal needs is something else again. Add to the 140,000 medicaments mentioned a while back the 129 standard laboratory procedures recently listed in a current hospital price sheet and you have a formidable foundation for the high cost of medical care or, what it often comes to, *redundant* medical care

Taken all together, what with the man-hours of the doctor and the woman-hours of the nurse, with board and lodging and the ancillary services appended, it approaches only the income tax for awesomeness.

As my 1956 reflection on this subject I beg leave to read into this record a clipping from a St. Louis paper of April 21 which I regard as the thumbnail sketch of the year. It is headed:

STUDENT LEARNS IT COST HIM $36.70 TO BE A HERO

It seems young Henry McN.. "swam 500 yards through cold, choppy waters last week to aid Fred P.., 12 years old, who was drifting out to sea in a rowboat. McN.. rowed the boat back to shore.

"Thoroughly chilled, he was held overnight at S.. Hospital for observation, and spent another day at home, resting up.

"The hospital bill arrived yesterday: $24.35 for room and board; $12.00 for laboratory fees and thirty-five cents for a prescription.

"A student at W.. Institute, McN.. says he doesn't have the money and he doesn't know where he can get it. He does odd jobs to support his wife and child."

I quote this merely to suggest that, in an era when prescriptions can still be had for thirty-five cents, all is not lost.

In a very thought-provoking article entitled, "Hazards of Modern Diagnosis and Therapy," David Barr, of Cornell, points out the dangers

of the indiscriminate use of new and unfamiliar drugs and especially the effects of modifying the internal environment by multiple diagnostic and therapeutic procedures simultaneously applied.

"A seriously ill patient," he writes, "or one who has been subjected to a major operation may receive twenty to forty different drugs in addition to numerous mechanical procedures. His management may actually require the use of anesthetics, sedatives, narcotics, antibiotics in variety, phenylephrine (neosynephrin) hydrochloride, arterenol, digitalis, diuretics, bishydroxy-coumarin, cortisone, transfusions, infusions and finally antihistaminics, either for the correction of symptoms of his disease or to combat the toxic manifestations of other drugs. Since such combinations of measures and medicines are frequent, it is not surprising that iatrogenic disturbances are frequent."

He reports that, in a period when approximately 1,000 patients were admitted to a large hospital, "more than fifty major toxic reactions and accidents were encountered." Some of these were the cause of hospitalization, others occurred in the wards.

He reflects that, in view of this five per cent of "unfortunate sequelae and accidents attributable to sanctioned and well-intentioned diagnosis," iatrogenic disease was one of the commonest conditions encountered in the period.

President Eisenhower was not talking specifically to us medical men of 1956, but he put his finger on the button when he observed that "the great genius is the man who can do the average thing when everybody else is going crazy."

* * *

SUMMARY AND CONCLUSIONS? This lecture is in itself a summary and I promised at the beginning only to put down what I saw, and let you and Bill Wherry draw your own conclusions.

Any attempt to epitomize this age in medicine, as indeed in almost everything else, would result in oversimplification, but perhaps it would not be too simple to suggest that the old ideal of healing the sick and giving them comfort as well as physic still has its points. What comfort to them, failing and afraid, to know as they sail past him on some production line, that the Big Man running the show invented a new virus and got his picture in the paper?

Souls and cells are somehow confused in this complex era characterized by curious paradoxes of prosperity, uncertainty, and, let's face it, fright: the highest standard of living and fear of inflation; the senseless chain-smoking and fear of cancer; double overtime, vacations with pay and fear of indigence; wonder-upon-wonder drugs and fear of viruses; overindulgence and fear of fat; the lengthening of life and fear of old age.

"I live all the time like a king," writes Don Herold, "whose enemies have threatened to poison him." The blessings of today and the brilliant promises of tomorrow seem not only too good to be true, but also too hard to bear.

Here in medicine there is a crying need for leadership to take us out of this nonsense; and let us lay the challenge right where it belongs—in the lap of the Professors. They alone can teach the coming doctor, in his formative years, the crashing difference between culture and mere knowledge; between leadership and mere prominence. They, and only they, by guidance and example can lift him above the great flood of technicians and inspire him to regain some of the lost dignity of his calling.

It requires a strong constitution to keep up with 1956.

I have just read somewhere that the latest wonder drug is so powerful you can't take it unless you are in perfect health.

XII

FACIAL PARALYSIS IN THE UVEOPAROTID FEVER OF BOECK'S SARCOID

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In 1909, Heerfordt[1] first described the syndrome characterized by uveitis, mild fever, enlargement of the parotid glands and paralysis of cranial nerves. Of the cranial nerves, the facial nerve is the most commonly involved. The similarity to tuberculosis led him to suggest that this represented a mild form of that disease. It remained, however, for Pautrier[2] in 1937 to relate the syndrome of Heerfordt to Boeck's sarcoid.

Parotid swelling is usually bilateral with moderate enlargement of the glands. Pain is not present, but dryness of the mouth is a common complaint. The parotitis subsides over a period of two weeks to several months. Rarely the submaxillary and sublingual glands are involved. In about 20 per cent, the lacrimal glands are involved.

The etiology of the disease remains unknown. The tubercle bacillus has been considered a possible etiological agent because the disease often terminates in tuberculosis. There has been, however, no convincing proof of the relationship. Clinically, the two diseases have similar symptomatology. The syndrome of Heerfordt is usually found in the second and third decade. The early symptoms include malaise, mild fever, gastro-intestinal disturbances, skin eruptions, joint pains and night sweats.

Bilateral uveitis is present in all cases previously described. Iridocyclitis is the most constant finding. Nodules in the iris are seen which closely resemble those of tuberculosis. Posterior synechiae often develop, as well as many other ocular manifestations.

Facial paralysis usually follows the parotitis from a few days to six months and is of sudden onset. The facial paralysis is of the peripheral type and usually resolves spontaneously. Colover[3] re-

Presented before the Pacific Coast Oto-Ophthalmological Society April 15, 1956.

viewed 115 cases with involvement of the nervous system. In about one-half of these, paralysis of the facial nerve was found. Levin[4] reported that in about 50 per cent of the cases of uveoparotid fever there is some involvement of the nervous system.

The cause of the facial paralysis has remained an enigma. Garland and Thomson[5] believe the facial paralysis is due to parotitis with pressure upon the nerve. They explain the presence of paralysis without parotid swelling as being due to swelling deep within the gland in the region of the stylomastoid foramen.

Colover, however, described a case in which the facial paralysis was improving although the parotid gland still showed enlargement. He pointed out that this would indicate that paralysis was not due to the involvement of the gland. In the literature there is rare mention of involvement of taste or hearing to indicate the position at which the nerve was involved. Colover, in the same paper, presented two cases in which taste was involved. In one of these, recovery from bilateral facial paralysis spontaneously occurred. This again would indicate that swelling of the parotid gland is not the etiological factor in involvement of the VII cranial nerve.

The lesions of Boeck's sarcoid have been found in almost every part of the nervous system and vary from chronic meningeal lesions to actual tumor-like masses. Reis and Tothfield[6] presented a case in which autopsy revealed infiltration of the optic nerves and olfactory bulb. Paralysis of the facial nerve had not occurred though it was felt that these findings could well explain the cranial nerve paralyses. Mazza[7] reported direct involvement of the nerve trunks with the formation of grossly visible nodules along the trunk, and Erickson[8] presented a single case in which sarcoid lesions were found within the brain. Ricker and Clard,[9] in a series of 22 autopsies, found the brain involved with sarcoid in three, and these took the form of miliary sarcoid lesions throughout.

The case to be presented here showed the findings typical of Heerfordt's syndrome with uveitis and facial paralysis. No parotid enlargement was noted, however, and the facial paralysis failed to show spontaneous regression.

REPORT OF A CASE

The patient is a 34 year old Negress, married and employed as a postal clerk. She is the mother of two children. Prodromal symptoms began in 1953 with joint pains. The pains recurred again fre-

quently in the spring of 1954, with resultant stiffness in the joints of the arms and legs. This was of mild severity and did not interfere with her job or housework. In August 1954 she noted blurring of vision in both eyes, but it was more severe on the left side.

On November 8 she found difficulty in moving the right side of her face while chewing. Following this, complete paralysis rapidly developed on that side. On November 15, in addition to the blurred vision, she had pain and redness of the left eye. Low grade fever was also present at this time, when she sought ophthalmological care.

On November 20, 1954, she was found to have bilateral granulomatous type anterior segment uveitis. Many large granulomata were on the endothelium of both cornea. The anterior chamber revealed mild flare on the left side with a few cells. On the right a moderately severe flare was present with many cells. No posterior segment disease was evident.

Physical examination revealed cervical lymphadenopathy. Cardiac findings were normal and the chest was clear on percussion and auscultation. The chest x-ray revealed bilateral hilar nodes and right paratracheal nodes. The lungs were clear. No parenchymal pulmonary disease was noted. An x-ray of the hands at the same time showed no evidence of sarcoid.

The patient was first seen in the Department of Otolaryngology on December 13, 1954. In addition to the history of facial weakness, she stated that tinnitus was present in the right ear and that sounds were disagreeably loud on that side. This began at the time of the facial paralysis. Her otolaryngological history was otherwise negative.

On examination, she was found to have a peripheral facial paralysis on the right side (Fig. 1). Salty, sweet and bitter materials could not be tasted on the right side. Her audiogram was within normal limits. X-rays revealed both mastoids to be well pneumatized and without evidence of destruction. Faradic stimulation failed to produce response on the affected side. A right scalene node biopsy was performed on December 18, 1954, and revealed multiple noncaseating, tiny granulomata. The pathological diagnosis was that of sarcoidosis.

Physiotherapy to the right face was begun and the patient was placed upon cortisone in high doses and followed with a maintenance dose of 50 mgm orally, three times a day. Optic findings improved greatly and on December 30 only one tiny residual granuloma was

Fig. 1.—Dec. 17, 1954. Pre-operative to facial paralysis on right.

noted in the right eye along the iris margin. No improvement at all was noted in the facial nerve.

The question now arose as to the advisability of surgical intervention to restore function in the facial nerve. A complete review of the literature failed to reveal surgical decompression having been performed in the presence of sarcoidosis. An attempt was made to correlate the length of duration of facial paralysis and the prognosis for return of function in the nerve.

Fifty-six reported cases of facial paralysis in well authenticated uveoparotid fever were found. Of these, only the reports included in the table made mention of the duration or the outcome of the facial paralysis.

It would appear that the course of the facial paralysis is not unlike that of idiopathic Bell's palsy. If evidence of improvement in the paralysis fails to appear early, the chances of its persisting permanently are great; however, among the authors who mentioned the course of the facial paralysis, it was stated that the paralysis rarely persisted, but recovery appeared within the period of a few weeks.

TABLE I

CASE NUMBER AND AUTHOR	PAROTITIS PRESENT	SIDE	LENGTH OF TIME OF PARALYSIS
1. Daireaux[10]	Yes	Right	One month
2. Heerfordt[1]	Yes	Right	Transient—time not given
3. Leeksma[11]	Yes	Not stated	Two weeks
4. Ramsay[12]	Yes	Bilateral	Three to four weeks
5. Feling and Viner[13]	Yes	Bilateral	Left, four weeks; right, less than 15 weeks
6. MacBride[14]	Yes	Bilateral	April to July
7. Garland and Thomson[5]	No	Bilateral	Recovered. Time not given.
8. Garland and Thomson[15]	Yes	Not given	September to August. Partial paralysis still present.
9. Garland and Thomson[5]	Yes	Not given	Recovered. Time not given.
10. Tanner[16]	Yes	Left	Four weeks
11. Tanner	Yes	Left	Four weeks
12. Tait[17]	Yes	Bilateral	Left, had cleared at five months; right, persisted
13. McCurray[18]	Yes	Left	Four weeks
14. Cogan[19]	Yes	Right	Two months
15. Folger[20]	Not stated	Left	Two to three weeks
16. Schupbach[21]	Yes	Right	Middle of May to July
17. Pautrier[2]	Yes	Left	Less than four months. Dates not given exactly.
18. Thompson[22]	Yes	Bilateral	Six months. Still present. —
19. Thompson	Yes	Not given	Three weeks
20. Thompson	Yes	Not given	Present one year later —
21. Thompson	Yes	Left	Cleared. Dates not given. Residual at seven months.
22. Lesne[23]	Yes	Bilateral	Cleared. Dates not given.
23. Palmer[24]	Yes	Not given	Four weeks
24. Reisner[25]	Yes	Not given	Resolved. Time not given.
25. Tepper[26]	Yes	Bilateral	June 28 to August 23
26. Curtis[27]	Questionable	Bilateral	April 7 to May 19
27. Lewis[28]	No	Bilateral	Clear in three weeks
28. Lewis[28]	No	Bilateral	Persisted 3 months. No further follow-up.
29. Ozazewski[29]	Yes	Right	"A few days"
30. Jefferson	Yes	Left	August 12 to October 15
31. Jefferson	Yes	Left	May 25 to August 3. Recurred with almost no recovery.
		Right	Less than one month

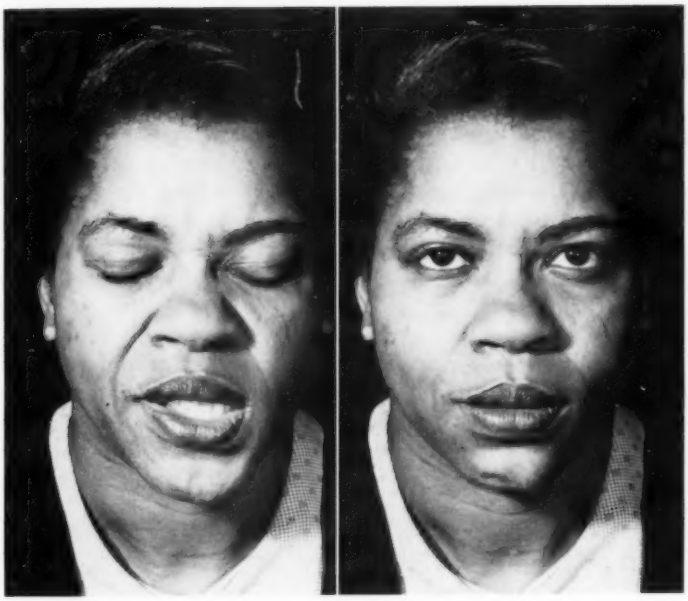

Fig. 2.—6 months post-operative showing full recovery of facial nerve.

Following the precepts of Kettel,[31] Sullivan,[32] Hilger[33] and Cawthorne,[34] it was felt that in the present case, decompression was advisable. Kettel proposed observation of the patient for a two-month period and surgical intervention if there is failure of the nerve to show signs of regeneration. The above review of the literature of uveoparotid fever would support this period of observation.

Decompression of the right facial nerve was performed on January 12, 1955. The patient was prepared in the usual fashion under endotracheal anesthesia. The Lempert incision as modified by Work for modified mastoidectomy was performed. The mastoid was found highly pneumatized. A few adhesions were noted about the head of the malleus and the incus, but the facial canal appeared intact. The nerve was uncapped in the transverse portion. It bulged quickly from the canal to three times its normal size. The entire facial canal was decompressed and throughout the mastoid course of the nerve it was found markedly edematous upon splitting the nerve sheath. The mastoid was completely exenterated. The flap was turned into position

and held in place with paraffin mesh gauze. The postoperative course was uneventful and by March 1, the nerve showed good improvement (Fig. 2). This has persisted to the present.

COMMENT

In summary, this appears to be the first case of reported uveoparotid fever in which decompression of the facial nerve was performed. The presence of sarcoidosis was confirmed by biopsy of a scalene node. Findings at the time of decompression were of marked edema of the facial nerve. Healing occurred promptly following decompression.

In conclusion, it may be implied that in sarcoidosis with paralysis of the facial nerve, the management should be similar to that normally employed for treatment of Bell's palsy with surgical intervention if the nerve fails to show regeneration. Certainly a single case does not present a mode of handling this problem, but it is hoped that this may be a guide to otologists confronted with this complex and poorly understood disease.

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XIII

THE FACIAL NERVE IN THE SURGERY OF CONGENITAL ATRESIA OF THE EAR

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Congenital atresia of the ear is due to the anomalous development of the posterior ends of the first and second branchial arches and the first branchial cleft and pouch. The facial nerve being the nerve of the second arch may be involved.

Henner and Buckingham[1] noted severe anomalies of the facial nerve pathway only in their group of severe anomalies with microtia, complete stenosis, sclerotic mastoid, severe deformity or absence of ossicles.

Kenneth and Sullivan[2] noted that in pneumatized mastoid the facial nerve was more or less normal.

Holmes[3] states that the abnormally placed facial nerve has been encountered three times, twice in a well pneumatized bone.

Kettel[10] reporting on 125 cases for the Ballance and Duel operation reports that in no less than seven cases more or less considerable deviation of the normal course could be demonstrated.

Surgery of atresia, because of the unpredicted anomalies that may be present, is like sailing in an uncharted sea. The fear of injury to the facial nerve dominates the surgical technique, and is still an argument against operation in unilateral involvement.

ANATOMY OF THE FACIAL NERVE IN THE TEMPORAL BONE

The labyrinthine segment is 4 mm in length. The nerve enters the bone through the internal auditory meatus from the posterior cranial fossa running from within outwards in the horizontal plane between the cochlea and vestibule, ending in the geniculate ganglion.

It then makes a right-angle turn at the genu, commencing the tympanic segment, (8 mm in length and 2.5 mm in diameter) enters

the fallopian canal on the internal wall of the middle ear just above and slightly anterior to the point where the ligament of the tensor tympani muscle is attached to the handle of the malleus. The tympanic segment runs directly backwards and slightly outwards through the fallopian canal in the horizontal plane until it passes between the horizontal canal and the oval window, then it makes another right-angle turn forming the mastoid segment.

The pyramidal segment, 2 to 6 mm in length, is the bend between the tympanic and mastoid segments. In the aditus the downward bend lies about 3 mm below the floor. The mastoid segment, 7 mm in length and 4 mm in diameter, runs directly downwards in the vertical plane along the bottom of the posterior canal walls and medial to the suture between the tympanic and mastoid parts of the temporal bone —the tympanomastoid suture. The facial nerve is not found posterior to the suture. It may be slightly anterior. It leaves the temporal bone through the stylomastoid foramen.

The chorda tympani in the infant leaves the facial nerve outside the skull, but in the adult enters the iter chorde approximately 4 mm above the stylomastoid foramen.

In this channel it runs almost directly upwards and forwards to the outer side of the posterior wall of the tympanum beside the rim of the tympanic membrane. It crosses the outer wall of the tympanic cavity enveloped in a fold of mucous membrane and passes medially to the manubrium of the malleus and above the insertion of the tensor tympanic muscle. It leaves the tympanic cavity by way of the iter chordae anterius.

DEVELOPMENT OF THE FACIAL CANAL

In late embryonic life and the first year of post embryonic life,[4] the facial nerve lies in a sulcus of the labyrinthic capsule which is closed to form a canal by the development of a bony cover on a connective tissue base.

The lowest portion of the facial canal is not formed in the above manner but originates together with certain parts of the posterior and inferior walls of the tympanic cavity and the sheath of the styloid process, from the very posterior part of the skeletal portion of the second branchial arch, the cartilagenous laterohyal, the laterotympanohyal. In normal circumstances the laterohyal forms the eminentia styloidea and participates in the formation of the lower part of the facial canal.[8]

ANOMALIES OF THE FACIAL NERVE AND CANAL IN THE TEMPORAL BONE

1. The facial nerve emerged from the temporal bone approximately 1 cm cephalad to the middle ear and the nerve was cut at the upper end of the endaural incision (Kinnley[5]).

2. The nerve instead of following the fallopian canal after the geniculate ganglion extended directly across the cavum tympani supported by coarse connective tissue. At the lateral boundary of the tympanic cavity, a small off-shoot of the nerve extended posteriorly. The latter gradually worked its way posteriorly and becoming encased in a bony canal, followed somewhat the course of the usual fourth portion of the VII nerve. The main part of the nerve after crossing the tympanic cavity becomes external by penetrating the connective tissue which replaced the region of the tympanic membrane (Hagens[6]). Fibrous tissue may seal the middle ear space and the nerve may be embedded in this tissue (Meltzer[7]).

3. The facial nerve took a course from the geniculate ganglion between the horizontal canal and stapes, then instead of going through the stylomastoid foramen it ran in a circle anteriorly, followed the location expected of the annulus tympanicus and made its exit from the bone apparently in the glenoid fossa (Holmes[3]).

4. The facial nerve after the geniculate ganglion turned backwards and was enclosed in the tegmen for a short distance. The tegmen showed an abnormally deep origin for the lateral labyrinthine wall going off just above the niche of the oval window. The facial nerve then entered the middle ear, lays for a short distance in an osseous canal on the under surface of the tegmen near its point of origin. The nerve then instead of going downwards and backwards over the posterior superior circumference of the oval window frame turned laterally. It was partially enclosed running in an incomplete bony canal on the crest of a bony ridge which originated at the posterior part of the petrosum. The ridge extended from this point in an oblique direction anteromedially from the lateral medial wall of the middle ear and divided it in two parts. The nerve left the middle ear at a point which was farther forward and higher than usual. Immediately after leaving the middle ear it gave to the chorda tympani (Altman[8]).

5. The facial nerve was normal in its diagonal course across the middle ear but on reaching a point just below the middle of the

horizontal canal, it followed the cortex of the external auditory canal wall and came out at the junction of the anterior and inferior portion of the wall instead of descending directly to the stylomastoid foramen (Schattner[4]).

6. The squama was situated very close to the oval window of the inner capsule so that the tympanic cavity was extremely low. In this bony bridge ran the ventrally displaced facial nerve.

The low tympanic cavity was so small that the head of the stapes touched the bony wall of the facial canal and was joined to it by connective tissues (Ruedi[9]).

7. The nerve in the posterior part of its short horizontal course, although it lay below the prominence of the lateral semicircular canal may come to lie lateral and then posterior to the posterior end of this canal, also just lateral to and above the ampullary end of the posterior ventrical canal.[14]

8. Part of the facial nerve descended directly in the usual course but another part, that which innervates the lower part of the face, swung backwards from the usual midpoint in the horizontal canal and did not descend until reaching the sinus wall which was perhaps one-half inch behind the posterior canal wall; on the top of the digastric groove it swung forwards again and joined the normal filament just above the stylomastoid foramen (Pierce[4]).

9. The diagonal course of the facial nerve in the middle ear was shortened; however, after making the first bend below the anterior border of the horizontal canal, the nerve did not descend directly but followed a more oblique course than usual. The nerve was therefore about three-eighths inch behind the posterior canal as it left the mastoid through a foramen, which was practically in the middle of the posterior part of the pastoid tip (Altman[4]).

10. Proximal to the chorda tympani the nerve formed a convex curve before resuming its course forwards and medially. The most prominent point of this posterior and convex curve is directly distal to the most overhanging part of the lateral semicircular canal (Kettel[10]).

11. The nerve was displaced 2 to 4 mm dorsally all along its vertical course; it did not pass along the posterior wall of the acoustic

meatus but medial to the threshold cells, between the posterior wall of the osseous meatus and the sinus sigmoideus (Kettel[10]).

In two of the three cases the nerve emerged from the anterior aspect of the mastoid process rather than from between the mastoid and syloid foramen.[14]

12. The nerve made a convex curve and was displaced backwards and medially (Kettel[10]).

13. The descending part of the facial nerve took a much more superficial course (Street[4]).

14. The nerve passed perpendicularly up to the dura instead of its usual bend under horizontal canal (Woodman[3]) (House[11]).

15. The facial nerve behind the oval window instead of going backwards and downwards, turned backwards, downwards and laterally at the crest of a ridge which protruded from the posterior wall of the middle ear into the cavity—which divided the lumen into a supralateral portion which communicated with the antrum and an inframedial portion which ended blindly. The facial nerve thus left the temporal bone more anteriorly than under normal circumstances. Its bony canal was also in this portion completely closed.

The pyramidal piece of bone which was intercalated between the posterior auditory process of the squama and the part of the petrous bone which forms the posterior portion of the floor of the middle ear participated in the formation of the lowermost part of the facial canal. The nerve ran between this bone and the petrous portion laterally and eventually left the temporal bone (Altman[12]).

16. The tympanic portion of the facial nerve ran in almost vertical direction with distortion of the medial wall of the middle ear (Kinney[5]).

17. The hypoplastic facial nerve went through the stapes between the crura in approximately the same way as the stapedial artery (Altman[4]).

18. The bulk of the fibres joined the chorda tympani leaving practically none to descend to the stylomastoid foramen (Dorothy Woolf[7]).

a) The chorda tympani nerve is rarely absent.

b) It may be found running outside the atresia plate in case this is formed by the hyperplastic upper end of Riechters' cartilage. The plate in this case does not occupy the site of the tympanic membrane but lies medial to it and that the tympanic cavity represents only the medial part of the normal tympanic cavity.[8]

c) It is found in the tympanic cavity inside the atresia plate and that indicates that the plate occupies the approximate site of the tympanic membrane.[12]

d) It may leave the facial nerve after this leaves the middle ear and runs on the outer side of the styloid.[8]

19. The facial nerve was extensively exposed without bony covering gaps in the osseous facial canal (Woolf[7]): 1) in part of the canal above oval window, 2) below the horizontal canal, 3) in the descending part.

The gap in the bony wall was bridged simply by ordinary fibrous connective tissue which was covered on the surface towards the middle ear cavity or towards the mastoid cells by epithelium normal to the region. Bilateral defects have been observed (Guild[13]).

REVIEW OF THE ANOMALIES WITH REGARD TO SURGICAL TECHNIQUE

1. The anomalous position of the facial nerve in the connective tissue outside the middle ear will indicate a retroauricular incision (Categories 1-3).

2. The varied and frequent abnormal positions in the mastoid do not recommend complete exenteration of the mastoid at least in cases 7 to 14.

3. The position of the nerve in the tegmen do not recommend direct atticotomy (Categories 4 and 6).

4. The area of bone in the mastoid with no record of facial nerve anomaly is in the angle bordered anteriorly by the line of fusion of the anterior border of the mastoid with the atresia plate; superiorly by the posterior root of the zygoma.

5. This site of election is utilized for:

a) The search for the antrum.

b) In the absence of antrum, as a route for entering the middle ear at the most posterior part of its lateral boundary after identifying the level of the tegmen.

c) This area allows ample bed for the skin graft.

6. The anomalous courses of the nerve in the tympanic cavity would recommend great care and avoiding much interference in the middle ear (Categories 2, 4, 14, 15, 16, 18, 19).

7. The anomalous position in relation with the lateral semicircular canal will show the difficulties, sometimes the impossibility of fenestration (Categories 7, 14).

8. Categories 2, 8, 17, 18 show that finding a facial canal does not exclude the presence of an anomalous facial nerve liable to injury in the middle ear or mastoid.

9. Category 18 directs the attention to great care for the chorda tympani nerve.

SURGICAL TECHNIQUE

Retroauricular Incision. The upper part of the mastoid is exposed to define the area of election in the angle between the line of fusion of the squama of the mastoid and the atresia plate anteriorly and the root of the zygoma superiorly.

Bone work is started here by gouge or burr taking care of a forward displaced sigmoid sinus in sclerotic mastoids, especially so on the right side.

Proceeding from behind forwards defining the tegmen mastoidi which gives an idea anteriorly about the level of the tegmen tympani. The depth in the search of the antrum can be gauged by the level of the atresia plate.

If the antrum is found, well and good. In case these is no antrum found—the ridge of bone between the formed bone cavity and the glenoid fossa is lowered down by burr below the level of the tegmen and the middle ear is entered at the most posterior part of its lateral border.

This ridge of bone is formed by the fusion of the mastoid and the atresia plate and suggests the line of the vertical descending mastoid segment of the facial nerve.

A small part of the atresia plate is removed by burr to allow examination of the middle ear and removal of ossicles.

Skin Graft. At the site of proposed meatus in complete atresia a rectangular skin flap with its base posteriorly is dissected superficially and reflected backwards to help in the formation of the postmeatal wall and to meet the skin graft in the bony cavity.

NOTE. Normally in search for the antrum the antral-tympano-eustachian axis with a slant of 30° downwards-forwards and 45° inwards is an absolute constant; the outer wall of the antrum must always be superficial to the plane of the tympanic membrane.[15]

This does not hold in atresia cases because of the variations of the position of the petrous part of the temporal bone and this, of course, is associated with a change in the position of the tegmen as well as other landmarks of importance.[4]

This technique was followed in the following two cases with sclerotic mastoids and facial abnormalities.

The atresia plate in both cases was completely osseous—passed medially—forwards from the mastoid border. Its line of fusion with the mastoid was vertical, meeting the posterior root of the zygoma at right angle.

REPORT OF CASES

CASE 1. S.A. Male, 23 years old. Both auricles were normal. There was a left complete atresia. The right meatus and drum were normal. There was conduction deafness with a 70 db loss in both ears. Bone conduction was good. X-ray showed a sclerotic mastoid, and a hypoplastic mandible.

Operation: 27-12-54. There was no antrum. A ridge of solid petrous bone was found passing vertically upwards carrying the facial nerve crossing the lateral semicircular canal. The posterior end of this was found displaced backwards and was fenestrated. The middle ear was hypoplastic. The incus was removed and the cavity lined with a Thiersch graft.

CASE 2. R.A.S., 10 years old, had a right microtia with complete stenosis. The left auricle, meatus and drum were normal. There was conduction deafness of 80 db loss in both ears. Bone conduction was good. Taste was normal. X-ray showed the right mastoid sclerotic. There was a mild degree of facial asymmetry and paral-

ysis of the right levator velum palatinum. There was underdevelopment of the right hand and leg and webbing of the neck.

Operation: 23-10-56. There was no antrum. The middle ear was hypoplastic; the lining mucous membrane was thick and pinkish. There were no ossicles, no muscles and no chorda tympani. A full thickness skin graft was obtained from the abdominal wall.

SUMMARY

The normal anatomy of the facial nerve and its recorded anomalies in the temporal bone are reviewed with the aim of elaborating a surgical approach for the surgery of congenital atresia of the ear founded on an anatomical basis.

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XIV

PROBLEMS IN THE IDENTIFICATION OF NON-ORGANIC HEARING LOSS

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The proper placement of children in schools for the deaf is dependent upon accurate identification of individuals with organic acoustic impairment. The task of diagnosing them is a highly complex one, since many disorders which show similar gross symptoms are sometimes confused with true deafness: i.e., mental retardation, non-organic loss of hearing, high frequency loss, conceptual and perceptual disturbances (often referred to under the less precise general term "aphasia"), brain injury, emotional disorders, mental illness, and others. Institutions established to train the deaf are not ordinarily equipped or staffed to handle students suffering from one of these ailments. For that reason, most schools for the deaf do not accept deaf children who are unable to attain an intelligence rating of 60 or 70 on reliable performance scales. They also reject aphasics and severely emotionally disturbed individuals, if the school lacks adequate facilities or personnel to justify their acceptance. Thus, the examining staff of such a school has a great responsibility in performing the type of differential diagnosis that will lead to placement in the most suitable educational program. It is widely recognized that the needs of children whose auditory deficit is of central origin are not being adequately met.

While the extent to which psychological hearing loss occurs has not been established by a large scale testing there is general agreement that it is more prevalent than formerly believed. The literature pertaining to the subject, however, is inconclusive. Ramsdell[3] stated that the number of psychologically deaf was larger than previously suspected but he presented no figures. Morrisett[2] estimated that 15 per cent of the cases of deafness recorded in any army testing center were of psychological origin. According to Shambaugh[4] in a study of

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3,120 children in public schools for the deaf the cause of deafness was probably psychogenic for 0.2 of 1 per cent of the group. Getz[1] recently pointed to the need for effective diagnosis, but gave no indication of the number or percentage of adequately diagnosed cases of non-organic hearing at the school where his study was done.

The practice of absorbing non-organically deaf, mentally deficient, or aphasic children into schools for the deaf is not advisable until these institutions become adequately equipped to handle these specialized problems. Every effort should be made to establish centers capable of training each exceptional group. The ideal program for the identifying of organically deaf children and eliminating other possible anomalies would involve the co-operation of several specialists, preferably functioning in a well co-ordinated center under medical supervision. Provision for the services of a certified otolaryngologist, a psychiatrist, a neurologist, a speech pathologist, an audiologist, and a psychologist skilled in the administration of nonverbal tests to the deaf should be made. It is fairly safe to assume that this ideal has not been completely achieved in most schools for the deaf. The individuals who accept responsibility for pre-entrance examination of deaf children may find it helpful to study the conditions that resemble functional hearing loss and sometimes lead to mistaken diagnosis.

SUBJECTS AND PROCEDURES

This study is based upon the pre-entrance psycho-acoustic examination of 521 prospective students at the California School for the Deaf, Riverside, California, during the school years from 1953 to 1956. The examinations were performed by one individual, a non-medical staff member, certified as a speech pathologist and psychologist and trained in audiology. The examiner was afforded the opportunity to study case history data and statements made during previous examinations. Also, he recorded statements made by teachers and supervising teachers which were in the nature of a diagnosis. A sample of the representative comments included the following: "probably psychogenic," "largely functional hearing loss," "emotional origin," "considerable functional overlay," "seems to be psychological or aphasic." During the three year period covered by this report, many of the students to whom these diagnoses referred were studied in 1) follow-up examinations, 2) observations of curricular and extracurricular behavior and 3) conferences with supervising and regular teachers.

Of the 521 prospective students who had been examined, 23 had been labelled "psychogenic" or functional cases according to the

information available to the authors. This figure is 4 per cent of the sample examined. Each of these individuals were studied according to these accepted audiological procedures: 1) a series of pure tone audiograms, 2) speech reception and noise perception tests, 3) noise interference level tests, 4) observations of reactions to gross sounds, 5) voice and speech diagnoses and 6) observation of psychological adjustment. Also, electroencephalographic and psychogalvanic skin resistance audiometry results, when obtainable, were studied. As a result of these studies to establish more definitely the existence of non-organic hearing loss, it was decided that 21 subjects showed other symptoms which were the primary etiological factors rather than functional loss of hearing. The symptoms or syndromes will be listed in order of their frequency.

1. *Mental Retardation.* Mental immaturity, as indicated by standardized nonverbal tests such as the Wechsler Intelligence Scale for Children, the Ontario School Ability Examination and the Grace Arthur Point Scale of Performance Tests, was the leading problem which had been overlooked by the person who had suspected functional loss of hearing. The writers found that, with the exception of a very small number of young deaf children, these tests were a valid measure of the native learning ability of hypacusic individuals. Seven of the suspected psychogenic cases were found to be mentally retarded or mentally deficient. Two or more of the performance scales were administered to these children and no great discrepancy between the scores was found.

2. *High Frequency Hearing Loss.* The child who has a high frequency hearing loss may have speech patterns somewhat like those of normal hearing children. He may respond to voices spoken with average loudness. While he may deduce the meaning of common expressions, he may not be able to repeat any words which contain a number of high frequency consonants such as th, f, s and t. If the hearing loss has been progressive, the individual may obtain the meaning by sound memory, hearing only segments of sound patterns once perceived completely. Many such children are suspected by their teachers of having a psychological hearing loss. Five examinees showed consistent high frequency losses but revealed none of the signs of functional hearing loss.

3. *Brain Injury.* Children who have sustained some degree of cerebral damage may have specific perceptual or conceptual deficiencies which interfere with the receptive or expressive phase of hearing

measurement. A slight brain injury might not be measurable by means of established medical procedures such as with the use of electroencephalographic, pneumoencephalographic and other neurological equipment. In the current study four subjects were found to have sufficient clinical evidence of brain damage to establish it as the cause for behavior patterns resembling those of the deaf. Non-organic hearing loss was ruled out because of consistent pure tone and speech audiometry responses along with normal behavior patterns.

4. *Functional Hearing Loss.* Hearing loss of a psychological nature remained a tenable diagnosis with three of the subjects so classified by enlightened people in their environment. This figure is one-half of one per cent of the sample. Only one of those three children remained at the school for a length of time adequate for study. He responded well to psychotherapy and expert classroom instruction and was subsequently transferred to the public schools. His present audiometric curves have consistently averaged 35 per cent higher than previous tests indicate.

5. *Serous Otitis Media.* The higher incidence of allergies in the region where this study was conducted doubtlessly accounted for several cases of serous otitis media. One of the subjects who was suspected of psychological hypacusis had shown several widely differing audiograms. In addition, he manifested withdrawal tendencies and insecurity. However, after an otolaryngological and psychological study it was determined that the variable audiograms and atypical behavior were the consequence of severe recurring allergic attacks, involving tubotympanitis and serous otitis media.

6. *Tinnitus.* In one subject tinnitus, which fluctuated greatly in severity and type, caused variable audiograms and a resultant presumption of psychogenesis as an etiological factor. This testing problem was solved with the use of warbled pure tone, which was not confused with the tinnitus.

7. *Negativism.* Some children do not speak in kindergarten for many weeks and may be suspected of having functional or organic hearing loss. One child in this investigation had not spoken in class and refused to respond in the testing situation during the first meeting. Several play therapy sessions were required to establish rapport and elicit speech. Audiometry showed him to have an average hearing loss of 45 decibels. After he had been fitted with a hearing aid and received sufficient auditory training, he was returned to the public schools.

Other problems which complicate the task of differential diagnosis include the early hearing fluctuations of otosclerosis, faulty mandibular articulation, and emotional disturbances. The possibility, also, that mental illness may be the cause of the auditory symptoms necessitates adequate psychological orientation to recognize the problem and the need for psychiatric referral. The problem of identifying functional loss of hearing is a highly specialized one, requiring participation of personnel trained in both psychology and audiology in close co-operation with or supervised by medical specialists. Safe minimum requirements might be stated as training equivalent to advanced audiological certification in the American Speech and Hearing Association and associate membership in the American Psychological Association. Only by using personnel who meet these requirements and follow recognized procedures under medical guidance for avoiding the foregoing erroneous diagnoses will the pre-entrance testing of deaf children be more effectively performed in schools for the deaf.

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XV

RHINOMANOMETRIC MEASUREMENTS OF THE NASAL PASSAGE

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Rhinomanometry is a method of utilizing the pressure variations in the rhinopharynx under normal respiration for measurement of the nasal passage. The method may be applied as anterior manometry of the pressure variations in the rhinopharynx via one of the nasal cavities or as posterior manometry of the pressure variations via a tube leading through the mouth and ending behind the soft palate.

Posterior rhinomanometry, first mentioned by Spiess[5] (1900), is the simpler method, as it directly indicates the pressure variations in the rhinopharynx during free respiration through both nasal cavities. On the other hand the tube behind the soft palate will often cause unpleasant sensations and unintended contractions and reflexes in the soft palate limiting the applicability of the method.

Anterior rhinomanometry described by Courtade[2] (1903) causes no inconvenience to the patient, but it is a partly unphysiological method because one-half of the nasal passage is closed during the measurement causing the respiration pressure to increase abnormally in the open nasal cavity. The posterior method has been employed to a very limited extent, the anterior by quite a number of investigators, usually by simply connecting the right and left nostril alternately to a U-tube liquid-manometer, the pressures indicated being considered a direct index of the permeability of the nasal cavity.

As might be expected this simplification will seldom yield to reproduceable results. The application of a recording manometer will show that the height of the pressure curves during respiration will

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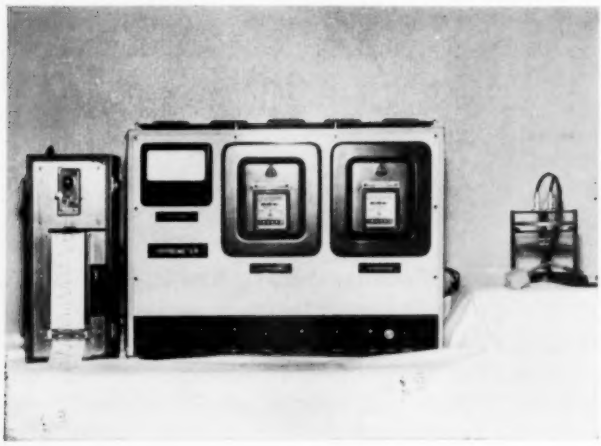

Fig. 1.—The rhinomanometer consisting (from right to left) of the electromechanic diaphragm-type manometer, the main-unit, containing the necessary electronic manometer circuits and the electronic integrator, and the recorder.

depend greatly on the frequency of respiration as well as on alterations in the amount of ventilation through the right and left cavity respectively.

In order to improve anterior manometry the authors have replaced the U-tube liquid-manometer with a recording diaphragm-type manometer, which is capable of integrating as well as recording inspiratory and expiratory pressure variations.

The rhinomanometer is composed of an electromechanic manometer with an attached recorder and electronic integration-unit.

The electromechanical manometer in outline corresponds with the so-called "Diaphragm-Type Micromanometer" described by Dibeler and Cordero[3] and Greenough and Williams.[4]

DESCRIPTION OF THE RHINOMANOMETER

The principle of the micromanometer mentioned is that of a corrugated diaphragm responding to pressure variations in the normal way, altering its distance to two air-core coils thereby causing a

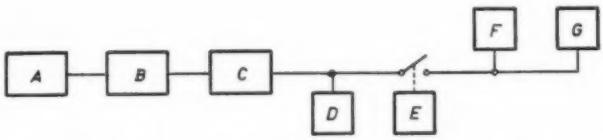

Fig. 2.—Block-diagram of the rhinomanometer: A. Pressure cell with diaphragm and coils; B. Electronic circuits attached to the coils; C. D.C. amplifier; D. Recorder; E. Timer; F. Rate-meter for expiration pressures; G. Rate-meter for inspiration pressures.

variation in the mutual inductance between these coils. This variation is utilized in the electronic circuits attached to the coils in such a way that a D.C. voltage proportional within wide limits to pressure variations is developed. Its polarity indicates whether the pressure measured is positive or negative.

The electronic integrator consists of a DC-amplifier designed along well-known lines and terminated by two rate-meters of the ampere-hour type, blocking of the rate-meters for opposite polarities of current results in one rate-meter summing up the expiration-pressures, and in the other one the inspiration pressures.

The rhinomanometer (Fig. 2) is equipped with a recorder and a timing-arrangement which, without being dependent on the pressure-indication proper, can switch on the rate-meters and switch them off again after a previously settled interval of time.

FORMULAS FOR CALCULATION OF NASAL RESISTANCE DURING RESPIRATION

Resistance of a Single Nasal Cavity. The volume (V) of air which during a certain time (T) passes a "conducting system" like a nasal cavity will be directly proportional to time, the linear average velocity (H) of the air-particles and to the cross-sectional area (L) of the cavity. This may be expressed by the equation:

$$V = H \times T \times L \quad (I)$$

Under identical conditions the linear average velocity is proportional to the driving pressure (P). Consequently it is permissible to substitute P for H in equation I giving:

$$V \sim P \times T \times L \quad (II)$$

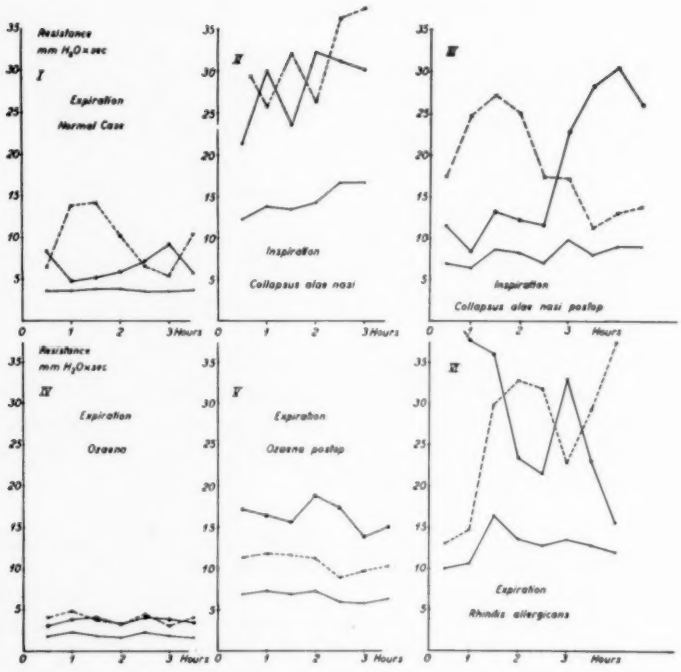

Fig. 3.—Resistance curves from normal person and from patients with various nasal diseases. o - o - o - o - o Resistance in right nasal cavity.
+ - - + - - + Resistance in left nasal cavity.
● - - ● - - ● - Resistance in right + left cavity.

I. Curves from a person with normal nasal passage and slight contraction of left cavity. The resistance in right and left side of the nose shows alternating oscillations, whereas the total nasal passage remains nearly constant.

II. Curves from a patient with collapsed alae nasi and greatly increased resistance during the inspiration phase.

III. The same patient after operation with lateral position of alae nasi a.m. Cinelli. Concha movements are clearly indicated by greatly varying resistance in the nasal cavities.

IV. Typical ozena-curves with pathologically low resistance values and very small variations in the resistance of the separate nasal cavities.

V. Curves from an ozena patient operated a.m. Eyries. A satisfactory reduction of the volume of the right nasal cavity has been obtained whereas the left cavity still appears too wide. The concha movements are very small in both nostrils indicating that a permanent injury to the conchastroma has taken place.

VI. Patient with rhinitis allergicans and greatly varying resistance produced by an increased and incoordinate activity of the turbinates.

The resistance (R) to passage of air will be inversely proportional to the cross-sectional area of the "conductor," so that $(\frac{1}{R})$ may with good approximation be substituted for (L) causing formula II to change to

$$V \times R \sim P \times T \quad \text{(III)}$$

Provided that the absolute volume of air passing the right or left nasal cavity is constant the formula III may be simplified to:

$$R \sim P \times T$$

For varying pressures the product $P \times T$ stands for the average pressure multiplied by the time used for inspiration and expiration respectively as summed up by the rate-meters of the integration-unit.

CALCULATION OF THE TOTAL RESISTANCE OF THE COMBINED NASAL CAVITIES

An evaluation of the total resistance of the combined nasal cavities based on knowledge of the resistance of the right and left cavities working separately may be performed in a very convenient way by comparison to an analogous electrical system of parallel conductors.

The legitimacy of transferring the conditions of conductivity and resistance in electrical conductors to conduction of air through tubes or cavities appears from the fact that in Ohm's law

$$I = \frac{E}{R}$$

the intensity of current (I) may be considered analogous to the amount of air volume per unit of time $(\frac{V}{T})$, the voltage (E) to the pressure (P) and the electrical resistance (R) to the resistance (R) to passage of air. Compare for instance formula III which may be written:

$$\frac{V}{T} \sim \frac{P}{R}$$

It is a well-known fact that a parallel combination of two electrical conductors R_1 and R_2 may be replaced by a single conductor the resistance R of which is determined by:

$$\frac{1}{R} = \frac{1}{R_1} + \frac{1}{R_2}$$

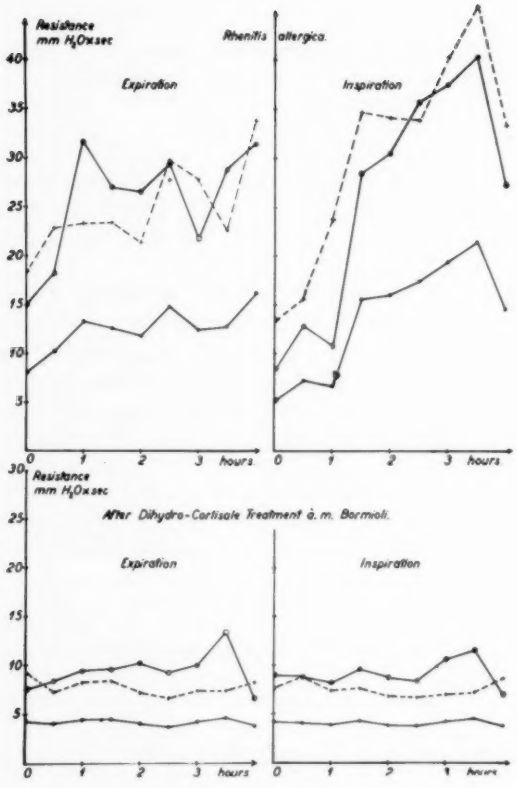

Fig. 4.—Resistance curves from a patient suffering from rhinitis allergicans before and after treatment with dihydro-cortisate a.m. Bormioli.[1]

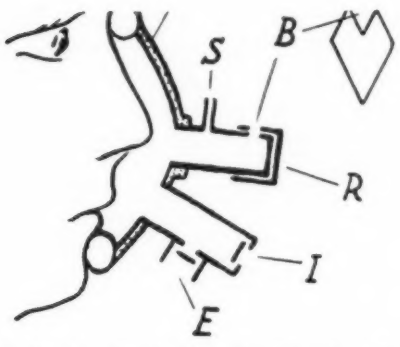

Fig. 5.—Application of mask with reduction-valve. R. Reduction valve consisting of two brass tubes equipped with rhombic openings. B. Diaphragm made up of the above mentioned rhombic openings. I and E. In- and expiration valves. S. Tube for connection of Rhinomanometer.

and transferred to the conditions of the nose we consequently arrive at the formula:

$$\frac{1}{P \times T} = \frac{1}{P_d \times T_d} + \frac{1}{P_s \times T_s}$$

where d and s refer to measurements in the right and left nasal cavity respectively.

CALCULATIONS OF VARIATIONS IN THE VENTILATION

Up till now we have used the same volume of ventilation for respiration through the right and left nasal cavity.

The right and left nasal cavity however have seldom the same width and resistance, which reacts upon the pulmonary ventilation with alterations in frequency and depth of respiration. The relation between pulmonary ventilation and alveolar ventilation may be expressed thus:

$$\text{Alveolar ventilation} = \text{pulmonary ventilation} - (\text{dead space} \times \text{frequency})$$

From this may be deducted, that by constant alveolar ventilation the (pulmonary) ventilation will decrease with decreasing respiration frequency and increase with increasing frequency. If an average frequency of 15, a respiratory volume of 500 cc and a dead space of

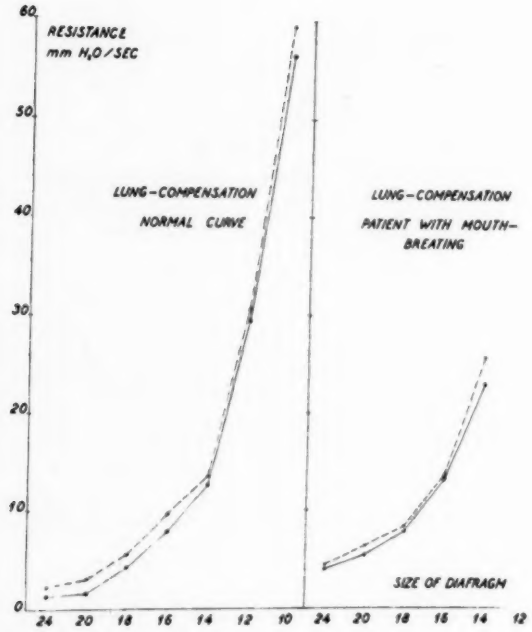

Fig. 6.—Diagram of respiration resistance, versus size of diaphragms indicating good (left) and bad lung-compensation (right).

150 cc is presumed the normal ventilation becomes 7500 cc per minute and the normal value of alveolar ventilation 5250 cc.

Every time the frequency is increased by 1 from 15, the ventilation will increase by 150 cc (the detrimental volume) or by two per cent. Consequently the measured product $P \times T$ which is proportionate to the amount of transported air (cf. eq. III) will increase by two per cent.

Consequently the product $P \times T$ must be corrected downwards by two percent for every "step" of frequency above 15 and similarly upwards by two percent for every "step" of frequency below 15. This correction is applied by multiplying the measured product by the factor

$$\frac{1}{1 + (F - 15) \times 0{,}02}$$

PRACTICAL MEASUREMENTS OF THE NASAL PASSAGE

The patient is placed conveniently in a chair and pressure variations are communicated from the right and left nostril respectively to the manometer, where they are converted to electrical qualities and the products of pressure and time of respiration for the two phases of respiration are summed up during a certain interval of time, normally one minute, by the two rate-meters, and the pressure-variations are at the same time indicated by a recorder from which the frequency may be estimated.

The values obtained are corrected by means of precalculated tables, so that one may easily arrive at the resistances R_d and R_s of the nasal cavities and at last also at the resistance (R) corresponding to free respiration through both nasal cavities.

The results are collected in a diagram with resistance along the ordinate and time along the abscissa. Measurements are taken every half hour and variations in the width or resistance of the nasal cavities as well as their influence on the permeability of the total nasal passage may be followed.

In order to have a fixed and reproduceable unit of nasal resistance the apparatus is adjusted by means of an Askani water manometer with mirror-reading. The pressure is measured in mm H_2O, time in seconds and resistance in mm H_2O by seconds.

PULMONARY COMPENSATION OF THE NASAL FUNCTION

For most normal persons the resistance to respiration through both nasal cavities is around 3-8 mm $H_2O \times$ second. Downwards there is a quite even transition to the atrophic rhinites, while the upper limit depends on the resistance which the lungs are able to conquer during respiration.

In order to get an impression of the ability of the lungs to compensate for increasing resistance a mask with a reduction-valve has been applied. The diaphragm of the reduction-valve is primarily adjusted to allow the patient to respire freely under both phases of respiration and afterwards it is gradually closed. The resistance during inspiration and expiration is read on the manometer.

Persons with good lung-compensation often show a prolonged inspiration at the expense of the expiration phase including a gradual

decrease of respiratory-frequency permitting a decrease of the ventilation even if the alveolar ventilation is kept at a constant level.

The experiences with lung compensation investigations are still rather limited but they seem to be able to supplement the common rhinomanometric measurements and give valuable information of the extent to which it is advisable to perform operations aiming at an expansion or a reduction of the nasal cavities.

SUMMARY

A method is described for measuring the resistance in the nasal cavities to pressure variations in the rhinopharynx when the right and left nasal cavities are alternately shut off. The measurements are made with a special apparatus, a rhinomanometer, which is capable of separating the two phases of respiration and summing up their pressure variations and of measuring the frequency of respiration.

From the values obtained—which are corrected according to the frequency by means of special formulas—it is possible to measure resistance deviations for the separate nasal cavity due to changes in the conditions of contractions of the conchae and calculate the total resistance in the nose under free respiration through both nasal cavities.

Diagrams indicate that abnormalities localized in the concha give typical resistance curves whereas those in the alae nasi cause increased resistance during the inspiration phase. Another diagram shows the effect of an operation of the nose with reduction of the lumina of the cavities.

A nasal stenosis may be compensated for by an exceptionally powerful pulmonary action, but respiration through the mouth may occur even with a normal nasal passage if the pulmonary function is reduced. Consequently it is necessary to supplement the measurement of the nasal passage by an appropriate investigation of the pulmonary function in which the patient respires against increasing resistance.

2 HAGEMANNSGADE

The work underlying this paper has been supported by grants from: P. Carl Petersen's Foundation; the Foundation of King Christian X of Denmark; and the rhinomanometer equipment has been furnished by the General Science Foundation of the State of Denmark.

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XVI

RESPIRATORY DISTRESS IN INFANCY

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While the occurrence of respiratory difficulty in new-born or young infants is infrequent, the recognition of its causes is important. These cases often present themselves as emergencies, and to deal with them the surgeon must have a comprehensive and systematic knowledge of their etiology. In many cases the measures necessary to correct them are simple and may be life-saving. Yet, in spite of this, the diagnosis is often only made at autopsy. This fact emphasizes the necessity for thorough understanding of the subject and also for closer co-operation between obstetrician, pediatrician and laryngologist.

Symptoms. These may begin in earliest infancy and the functions of respiration, phonation and deglutition may be interfered with singly or in combination. The obstetrician may notice strong respiratory efforts, with no evidence of air exchange. The cry may be absent, weak, hoarse or have a peculiar, muffled pharyngeal quality. There may be inspiratory or expiratory stridor. Inspiratory retraction may be suprasternal, supra- and infraclavicular and intercostal, or xiphoid and infracostal. Dyspnea is the most alarming symptom and may be punctuated by intermittent attacks of complete respiratory obstruction with cyanosis sometimes ending fatally.

Regurgitation, dysphagia and inability to suck are frequent and are accompanied by weight loss or failure to gain weight at a normal rate. Special positions of the patient may bring about partial relief from the above symptoms.

Examination. While in exceptional cases there may be no time to perform a full examination, the usual case permits a thorough one. A good light is essential to detect a cyanotic or ashy complexion. A

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decision must be made as to whether the infant is breathing through its nose or its mouth. If in doubt, a rubber catheter should be passed through the nose to ascertain the latter's patency. The mouth should be examined for receding chin, falling back of tongue and cleft palate. A thorough inspection and palpation of the neck is then conducted, while the head is flexed, extended and then turned to the right and left sides. This cannot be too strongly emphasized as large cervical masses are known to have been missed when this part of the examination had not been meticulously performed. Especially is this true of infants with short adipose necks, where the chin rests in the suprasternal notch and completely hides the neck from view.

Direct laryngoscopy is the only manner of inspecting the infant larynx and is therefore the most important single diagnostic procedure. This procedure may precipitate complete respiratory obstruction and therefore the surgeon must be prepared to perform a tracheotomy if necessary at the time of examination.

When examination of the upper respiratory passages does not disclose the cause of the obstruction, a bronchoscopy is performed. Disturbances of deglutition associated with respiratory obstruction indicate an esophagoscopy.

The diagnostic routine should also include x-ray studies of the chest and neck, particularly the lateral x-ray of the neck with the arms down and back and the head up and forward to obtain as much visualization of the trachea as possible. A fluoroscopic study of the esophagus with barium or lipiodol is also indicated to rule out compressing vascular anomalies or other anomalies of the tracheobronchial tree. Angiograms and surgical exploration (thoracotomy) may be necessary to clarify obscure conditions.

Causes. The following is an outline of conditions causing respiratory difficulty at or soon after birth:

A. Above the larynx.

1. Imperforate nares.
2. Stenosis or webs obstructing anterior nares.
3. Atresia of the posterior choanae.

These three conditions are rare and should be bilateral to cause dyspnea, cyanosis and difficulty in nursing or feeding.

4. Because infants have a very strong tendency to breathe through the nose, an abscess of the septum may give severe symptoms and even necessitate a tracheotomy to avoid asphyxia. A case of impending asphyxia due to nasal blockage caused by "rebound" swelling following the use of vasoconstrictor nose drops has been reported.[1]

5. Pierre Robin Syndrome: a) glossoptosis, b) micrognathia, c) cleft palate. The falling back of the tongue into the hypopharynx can cause great difficulty in breathing and swallowing. Aspiration pneumonitis and atelectasis further complicate the picture.

Severe examples of this condition are uncommon but mild cases are rather frequent. The condition is recognized at birth. In the following case the jaw was normal at birth but micrognathia developed after several months, a condition not hitherto reported.

REPORT OF A CASE

H.J. (Case No. 10416), a male infant born normally on February 26, 1956. He was admitted to the American University Hospital on April 15, 1956, with diarrhea of six days' duration. Examination revealed a normally developed child and under appropriate treatment he recovered and was discharged.

He was readmitted six months later with a history of noisy breathing, difficulty in swallowing and loss of weight of eight weeks' duration. Examination showed severe micrognathia and marked emaciation (Fig. 1). He weighed only 2500 gm. There was marked sternal retraction with inspiration and a constant and rather alarming stridor. These diminished when the infant was placed face downward, allowing the jaw and tongue to fall forward. Feeding was difficult and was interrupted by regurgitation, spitting and periods of respiratory distress. Orthostatic treatment, as suggested by Pierre Robin, failed to control the condition or bring about an increase in his weight; so on November 21, 1956, the tongue was sutured to the lower lip (Shukowsky-Douglas operation).[2,3] Respiratory symptoms were relieved at once. To date he is still in hospital.

6. Nasopharyngeal tumors may cause asphyxia neonatorum. They present the problem of obstruction to both nasal breathing and swallowing and attacks of cyanosis. Choristomas,[4] teratomas[5,6] and parasitic twins arising from the nasopharynx[7] have been described.

7. Thyroglossal cyst or lingual thyroid may be present at birth and may cause marked obstruction of respiration and deglutition.[8]

8. Congenital cysts of the base of the tongue have been described.[9]

Fig. 1.—Severe micrognathia and glossoptotic emaciation. Note sternal retraction. (See text.)

In the following case a large tongue associated with cerebral agenesis caused marked respiratory difficulty and cyanosis by obstructing the laryngeal inlet. To obviate this difficulty the tongue was kept permanently protruded.

REPORT OF A CASE

A five months old female infant (No. 104129) was admitted to the American University Hospital on April 18, 1956, with a history of attacks of choking, cyanosis and regurgitation on attempted feeding, since birth. The baby was a full-term normal delivery and was one of three healthy siblings. Examination disclosed microcephaly and micrognathia and an unusual amount of mucus in the nose and throat. The gag reflex was sluggish. There was generalized hypotonia. The tongue was large and protruded constantly from the mouth for about one inch. Attempts to depress the tongue for examining the throat resulted in cessation of respiration and cyanosis.

A lipiodol sip, in addition to showing a normal esophagus, resulted in a perfect bronchogram denoting dysfunction of the glottic mechanism. X-ray of the lungs showed consolidation and collapse of the right upper lobe but this soon responded to antibiotics.

Pneumoencephalography showed generalized symmetrical dilatation of the ventricular system and diminished cortex mainly on the right side. The diagnosis was cerebral atrophy.

The baby was discharged with a feeding tube to be cared for by his parents.

B. Obstruction in the larynx.

1. Congenital laryngeal stridor.

2. Congenital laryngeal webs.

These conditions produce inspiratory stridor and retractions and difficult feeding usually after the first day or two of life, but may be present at birth. Direct laryngoscopy establishes the diagnosis.

3. Congenital laryngeal atresia. This is obviously incompatible with life. Jackson and Jackson,[10] however, reported a case which was recognized at birth and the larynx forced open by pushing a bronchoscope through it into the trachea and establishing normal breathing.

A similar case was reported by Holinger et al.[11]

4. Congenital subglottic stenosis and structural anomalies of the cricoid cartilage.

Although the airway is often only slightly reduced in these conditions, the least laryngeal inflammation precipitates a tracheotomy since the limiting cricoid cartilage does not permit swelling of tissues in any direction other than inwardly, at the expense of the airway.

5. Congenital cysts of the larynx. These originate in the neighborhood of the ventricle and bulge into the glottic lumen producing a muffled or absent cry and respiratory obstruction.

6. Trauma by the aspirating catheter of the obstetrician may cause swelling or hematoma of the glottis resulting in a hoarse cry and respiratory difficulty.

7. Vocal cord paralysis may be associated with intracranial damage or pathologic conditions present at birth, viz., cerebral agenesis, cervical meningomyelitis or extensive cardiovascular anomalies involving the left recurrent laryngeal nerve. Bilateral abductor paralysis may occur at birth and can be fatal in a short time if not diagnosed immediately.

8. Tumors of the larynx are very rare in infants. A congenital hemangioma produces respiratory obstruction.[12]

C. Obstruction of the air passages below the laryngeal level.

1. Anomalies of the trachea comprise: a) agenesis and atresia, b) fibrous strictures or webs, c) stenosis or inspiratory collapse due to absence or deformity of tracheal cartilages, tracheoesophageal fistulae or anomalous vascular compression, d) tracheoesophageal lung.

Although much rarer than laryngeal anomalies, these conditions should be borne in mind when dealing with respiratory obstruction in infants. Progress in thoracic surgery has made many of these conditions amenable to treatment.

2. Cervical compression of the trachea may be due to dermoid cysts, cystic hygroma, thyroid cysts, aberrant thyroid tissue or branchial cysts.

REPORT OF A CASE

A 12 day old male infant (No. 84427) was first examined in the pediatric service at the American University Hospital on October 20, 1954. He was said to have had rapid stridorous breathing with cyanosis since birth. These symptoms became more marked with feeding and minimal when the head was extended.

The child was born at full term, the birth weight being 4 kg. His symptoms suggested pulmonary infection and he was treated with penicillin injections prior to his admission.

Physical examination revealed a well-developed male infant with stridor, subdiaphragmatic and intercostal inspiratory retractions and a moderate degree of cyanosis. These symptoms increased markedly on flexing the neck. The temperature was 37.5 degrees C rectally, pulse 130, respiration 60 per minute. The cry was weak but not hoarse. On casual inspection the neck looked normal but when the latter was extended and the face turned to the right a swelling could be seen on the left side which felt soft and cystic and measured about 3 cm in diameter. The remainder of the physical examination was negative.

A reduction in size of the cervical mass the following day suggested the diagnosis of branchial cyst.

Under ether and oxygen insufflation anesthesia the cyst was excised by Dr. J. McDonald. The following day a hematoma developed at the operative site causing marked respiratory obstruction. It was evacuated. The child had no further respiratory difficulty and was discharged on the fourteenth postoperative day.

The pathologist's report was as follows: Specimen consists of a cystic mass measuring 3.5 x 2.5 cm. It is lined by squamous epithelium and is rich in lymphoid tissue. It shows foci of vascular granulation tissue with polymorphonuclear infiltration. Diagnosis: "Branchial cyst."

Enlargement of the thymus gland is rare and is too frequently blamed for obstructive symptoms. If the gradual relief of self-limiting congenital anomalies such as laryngomalacia follows thymic irradiation, it is then incorrectly concluded that the thymus was to blame.

3. Mediastinal compression of the trachea may be caused by double arch of the aorta and transposition or abnormal course of great vessels. These often cause cyanosis, retractions and stridor in the first few months of life. Lipiodol esophagograms help in establishing the diagnosis.

4. Anomalies of the bronchi and lungs. The following may produce respiratory distress and cyanosis and are frequently confused with laryngeal obstruction.

a. Agenesis of the lung.

b. Bronchial compression due to anomalies of the heart and great vessels.

The following is an illustrative case.

REPORT OF A CASE

T.M., a five day old male infant (No. 90011), was admitted to the pediatric service of the American University Hospital on March 8, 1955, with the complaint of inability to take food by mouth due to harelip and cleft palate. He was a first child and there had been a full-term normal delivery. He cried immediately at birth but remained cyanosed for 12 days. There was no family history of disease or consanguinity. In the first trimester the mother had severe morning sickness.

Examination revealed right harelip and complete cleft palate. Both ear pinnas were deformed. The neck showed pterygium and a low hair line. There was hypertrichosis of the sacral region. The extremities showed axillary pterygium, webbing of the fingers and cubitus vulgus. The testes were undescended. The lungs were resonant. The cardiac rhythm was irregular but there were no murmurs.

The lipiodol esophagogram was normal and there was no evidence of a hiatus hernia.

X-ray of the chest showed hypertranslucency and diminished vascular markings. The left lower lobe was partially expanded. There was a bootlike configuration of the heart with elevation of the apex and a slight increase in size. Oblique views showed a right ventricular enlargement and on the posterior-anterior view there was a concavity in the region of the pulmonary conus.

The findings suggested the tetralogy of Fallot.

Electrocardiographic studies showed ventricular premature systoles and possible right ventricular enlargement.

Feeding by mouth always resulted in attacks of choking and cyanosis. He was therefore fed through a polyethylene tube but nevertheless was repeatedly subjected

to choking and cyanosis while crying or vomiting. The mouth had to be aspirated repeatedly during such attacks and artificial respiration was resorted to on several occasions.

In spite of careful feeding, administration of vitamins and antibiotics to combat aspiration pneumonia, his weight remained constant (2500 gm). He died 75 days after admission following a severe attack of cyanosis.

In addition to the external deformities already mentioned, autopsy revealed a) high interventricular septal defect, b) overriding of the aorta, c) hypertrophy of right ventricle, d) hypoplastic left ventricle, e) patent foramen ovale, f) absence of the main pulmonary artery, g) anomalous origin of pulmonary arteries from the aorta, h) absence of anastomosing branch between right and left precardinal venous system, i) persistence of proximal segment of the left precardinal venous system, j) anomalous insertion of right pulmonary vein into the right auricle.

These multiple anomalies of the heart and great vessels undoubtedly caused mediastinal bronchial compression and accounted for the respiratory distress in this infant.

c. Bronchogenic cysts and diverticulae.

d. Bronchoesophageal fistula where lower segment of esophagus is attached to the left bronchus rather than the trachea.

e. Bronchopleural fistula causing tension pneumothorax and extreme respiratory urgency.

f. Pulmonary cysts due to staphylococcic pneumonia may develop postnatally and simulate congenital cysts.

These conditions are of great clinical importance because they are responsible for some of the respiratory emergencies that occur in infants. They give rise either to atelectasis or to rapidly progressive emphysema through a check-valve mechanism producing a mediastinal shift and compressing other thoracic contents to the point of complete loss of function.

D. Rare conditions producing respiratory distress.

1. Idiopathic emphysema of a pulmonary lobe.[13]

2. Atelectasis of the newborn, whether due to nonexpansion or bronchial obstruction, causes dyspnea and cyanosis. Retractions, however, are of the xiphoid and infracostal areas and there is no stridor. A serious type of atelectasis due to congenital "flail chest" and previously known as "pulmonary hyaline membrane with resorption atelectasis"[7] may set in a few hours after birth. Paradoxic inspiratory retraction of the sternum is pathognomonic. Sternal traction is curative.

3. Diaphragmatic hernia may cause dyspnea, cyanosis and even noisy breathing that are sometimes confused with laryngeal obstruction.

4. Bronchiolitis. This produces respiratory distress and should be recognized by the laryngologist as tracheotomy and aspiration is often indicated as an emergency procedure.

5. Congenital dysphagia, due to neurogenic disorders of the esophagus may cause extreme respiratory urgency during feeding. Cerebral agenesis, the Riley-Day syndrome, familial bulbar paralysis and amyotonia congenita are some of the disorders responsible for this condition.[14]

REPORT OF A CASE

A.K. (No. 86878), a nine month old male, was first admitted to the pediatric service of the American University Hospital on September 27, 1955. He had never been able to swallow properly nor to breath well because of an overabundance of thick mucus in the throat.

He required tube feedings practically all his life, as attempts at feeding resulted in choking, cyanosis and regurgitation through the nose and mouth. He had frequent episodes of respiratory infection with fever, cough and cyanosis lasting a few days. These attacks responded to penicillin. The child was a full-term normal delivery.

Examination revealed severe mental and physical retardation. There was microcephaly and micrognathia. Saliva drooled from his mouth and nose. The gag reflex was present. There was generalized hypotonia.

Chest x-rays revealed atelectasis of the upper and middle right lobes but this responded to penicillin. Feedings attempted by mouth, however, were always aspirated and resulted in atelectasis.

Pneumoencephalography showed marked widening of the sulci and symmetrical dilatation of the ventricular system consistent with cerebral agenesis.

At direct laryngoscopy some difficulty was encountered in exposing the larynx because of micrognathia, but the larynx proper was perfectly normal. Esophagoscopy revealed no evidence of abnormality.

The child's course in hospital was unchanged. He was discharged unimproved. The parents were instructed to continue tube feeding.

SUMMARY AND CONCLUSIONS

1. A brief review of the causes, signs and symptoms of respiratory obstruction in infants is presented.

2. Illustrative cases are reported.

3. The role of the laryngologist in these cases is as follows:

a. He should know the etiology of respiratory distress in infants.

b. He should outline the investigation.

c. He remains captain until the diagnosis is made and the patient referred for treatment.

Merely referring these cases is an evasion of duty. The obstetrician, pediatrician and thoracic surgeon are of necessity and training ignorant of the diverse causes of respiratory difficulties. While each consultant is pre-eminent in his own field, the basic problem is an otolaryngological one. The otolaryngologist should direct the investigation, collect and correlate data and finally refer the case to the proper consultant.

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XVII

THE RHINOPLASTIC SURGEON AND THE POSSIBILITY OF LAWSUIT

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All plastic surgeons occasionally have the experience with dissatisfied patients who complain because the result of their operation is not what they had anticipated. This is most frequent possibly in operations about the face, and especially in relation to esthetic correction of a malformed nose, which the surgeon is asked to improve. The prominent position of the nose makes it an easy target for both accidental and intentional injury.

The greatest liability to the surgeon is in those cases in which the patient holds him responsible for the dissatisfaction, although the outcome may have been clearly discussed before the operation in case there was any reason for doubt about it. There are also those instances where, through negligence, or accidental injury, the operative area may have become disturbed while still in the early postoperative period, and have caused distortion of the otherwise satisfactory reconstruction.

Some individuals are inclined to make others responsible for their own faults and shortcomings, and often take recourse to law to relieve their feelings, and try to prove themselves right, even though they know they are wrong. Together with this attitude, the writer has observed that there are irresponsible, and sometimes actually "shyster" lawyers, who are willing to uphold the complaint of the dissatisfied patient, regardless of clear and demonstrable facts to the contrary.

Patients may be themselves irresponsible, and lack the character and integrity to consider the situation honestly; they may even resort to determined attempts to deceive in accounting for the cause of their complaint.

Any person who shows any signs of being psychotic should not be accepted as a patient for such surgery without the written request

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of an accredited psychiatrist. Such a person is apt to be dissatisfied with the result of any operation, however successful it may be from the surgeon's standpoint. Such a patient's dissatisfaction may not be limited to the results of the operation only, but may be projected upon the physician in the form of ideas of persecution, or otherwise.

The following case histories, from actual experience, illustrate briefly the type of patients who may be inclined to resort to legal process after surgery:

REPORT OF CASES

CASE 1. A young married woman, 27 years of age, was dissatisfied with her nose, which she considered very unattractive, and wished to have altered. A complete rhinoplasty was done, and the result was entirely satisfactory—both to her, and from the surgical standpoint. Three weeks later she returned, complaining that her nose now had an objectionable bulbous tip, for which she insisted that she knew no reason, although the physician suspected injury, which she definitely denied. Some days later, her husband came to the office with her, and when the question was casually asked as to how the present state of the nose could have come about, he gave the situation away by frankly saying that one night as he turned over in bed he had hit his wife's nose with his elbow. There had been considerable bleeding, but instead of calling the doctor promptly, she had waited for a week before visiting his office. The result was a separation of the newly operated tip cartilages, with extravasation of blood into the surrounding tissues.

CASE 2. A young woman with an unattractive nose underwent an operation for corrective cosmetic surgery, with excellent results. After four weeks' delay, she returned for inspection, complaining that the operation had failed, which appeared to be true. She was exceedingly unpleasant about the situation, and insisted that the surgeon was responsible for the condition. The situation was cleared up by x-ray examination, which revealed fracture of the nasal bones that could not have been caused during the operation. The findings were then explained to her, with the statement that there must have been an injury; further that the surgeon would repair the nose if she would tell him the exact truth. She then revealed that she had gone to a nightclub, where she had danced, and while doing so, someone struck at her partner, who had quickly avoided the blow, and the attacker's fist had struck her on the nose. The second operation was finally done, and the truth being admitted, the patient paid for it.

CASE 3. A young man, 26 years old, came for correction of a markedly deviated nose. A complete rhinoplasty was done, and the

nose successfully straightened. His family and his friends were thoroughly satisfied and pleased with the cosmetic result, but the patient himself was not. His nose seemed strange to him and he did not feel like himself, so he wished to have his twisted nose restored by a second operation. He was advised that this would be more difficult than the first one, and satisfactory results could not be promised him. He was advised to see a psychiatrist, but this only served to add to his disturbed feelings. He was not seen then for six months, and when he returned, his nose was crooked again. His story was that he had been in a fight, and by a blow upon it his nose had been misshapen again. But even then he was not satisfied, and wished another operation to straighten it once more. This request was refused.

The plastic surgeon, particularly in doing cosmetic operations, should always use great care in speaking about possible lack of success before a patient, if he intends to operate. All of his assistants should also be aware of the need for such caution, to avoid an experience of this writer: while during an operation, a surgical assistant quite openly asked the doctor how many suits for malpractice he had had. A patient who is being operated upon with only local anesthesia necessarily hears all the conversation that goes on among the personnel at the operation.

In the interest of professional safety, it is the writer's custom to keep a very detailed record of any injury, however slight it may be, since the patient's memory and word cannot be depended upon.

To insure as great safety as possible from this viewpoint, the writer requires a signed request for the operation, which includes the following paragraph:

"I *understand and agree* that in rendering such treatment to me, Dr. Seltzer undertakes to exercise his best skill and judgment as a physician and surgeon, but does not warrant, guarantee, or make any special contract or promises with respect to the result of this operation, or treatment which shall be given me."

This agreement is signed both by the patient and by the operating surgeon, with a witness' signature also.

These cases illustrate the potential difficulties which may lead to involvement with the law, although, fortunately, there was no such outcome here. However, such situations, if not handled tactfully, do at times come to legal action.

2104 Spruce Street

Society Proceedings

ST. LOUIS NOSE AND THROAT CLUB

Meeting of Wednesday, March 20, 1957

THE PRESIDENT, DR. CHARLES E. EIMER, IN THE CHAIR

XVIII

Side Effects of Drugs Used for Hypertension

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ST. LOUIS, MO.

Antihypertensive drugs are being used increasingly. Control of chronic arterial hypertension has improved greatly in the last five years, and powerful new drugs have been developed. Today there are agents which are capable of reducing or controlling most hypertensive patients, providing the drugs can be tolerated in adequate amounts. Undesirable side effects are common to all of these drugs, and the reduction of, or the suppression of, pressure in itself is not without risk.

The successful control of hypertension cannot be accomplished by haphazard or intermittent therapy. Once begun these drugs must be given in adequate doses at regular intervals; side effects are to be prevented before they occur, or are to be treated as they arise. A physician completely familiar with the primary effect and the side actions of these drugs is essential; an intelligent patient educated also in the use of these drugs and aware of the side effects is another essential to successful control. The otolaryngologist must be aware of these problems, for he is going to be consulted by patients already on antihypertensive programs, or who should be on therapy without delay.

Briefly, the pathogenesis of most hypertension may be considered a result of neurogenic and nephrogenic factors. The neurogenic influ-

ence through the sympathetic portion of the autonomic nervous system produces intermittent, generalized vasospasm. The intermittent neurogenic vasospasm in time becomes converted to chronic sustained vasospasm probably under the influence of a nephrogenic vasoconstrictor substance. When two different factors are producing hypertension, two different drugs may be required for combined reversal of the vasospasm. When two or more agents are required for control, the possibility of side effects is enhanced. Drugs are available that act upon autonomic ganglia and upon sympathetic areas of the brain; another drug acts directly on vascular smooth muscle, and has the capacity to inactivate known pressor substances important in hypertension. To some degree in many cases of chronic hypertension, sodium ions plus sodium-retaining steroids influence vasospasm. The invariable renal ischemia accompanying chronic hypertension is associated with urinary loss of sodium chloride; it is believed that the adrenal cortex is thereby influenced to produce more salt-retaining steroid hormones than ordinarily.

Of the drugs acting directly or indirectly on the central sympathetic areas, rauwolfia alkaloids are the most generously used. Chlorpromazine (Thorazine) is thought to affect the vasomotor center in some manner. Although large doses produce some fall in blood pressure, it has many side effects, and is of little antihypertensive value except indirectly through its sedative action. Protoveratrine alkaloids from veratrum stimulate carotid sinus and aortic depressor nerves causing increased depressor activity, and side effects of vagal stimulation with a narrow margin between therapeutic and toxic dosage. The rapid appearance of tolerance prevents maintenance of normotension with this drug alone.

Because rauwolfia alkaloids, and particularly reserpine, are mildly hypotensive, there has been a common tendency to employ them freely and without close observation. Their effect is a result of decreased sympathetic outflow and relative parasympathetic over-activity. Therefore, vasospasm from nervous reactions is partially inhibited, but side effects are frequent and numerous. Undesirable consequences resulting from over-activity of parasympathetics include bradycardia, gastric hyperacidity, gastrointestinal hypermotility sometimes to the point of diarrhea, activitation of peptic ulcers, and initiation of ulcerative colitis. Bradycardia, extra-systoles, auricular fibrillation, palpitation, paroxysmal arrhythmia, alarming chest pain, dyspnea, and near shock occur. The bradycardia of rauwolfia may lead to inadequate administration of digitalis by distorting the usual guides for simul-

taneous therapy. Effects similar to a prefrontal lobotomy include loss of ambition, inability to adjust and inability to meet new situations. Increased appetite and slowing of physical activity may lead to a subtle true gain in weight. Occasional marked fluid retention with edema and dyspnea has occurred within a week of initiating rauwolfia treatment, and this might lead to an erroneous supposition of myocardial insufficiency. Nasal stuffiness, epistaxis, and headaches are not uncommon. More serious late side effects include unusual emotional depression, anxiety, nervousness, insomnia, nightmares, and agitated depressive psychosis with suicidal tendencies.

Hydralazine (Apresoline) acts directly on vascular smooth muscle, and is often added to the milder agents when hypertension is resistant. It also impairs the action of histaminase, and headache has been produced or aggravated in almost one-third of patients so treated. Flushing, excessive lacrimation, nasal obstruction, anorexia and nausea have been almost as common. Histamine provoked hyperacidity seems to have produced hemorrhage from peptic ulcers. Chemosis and tachycardia occur. Angina pectoris and occasional infarction seem related to Apresoline. In 5 to 10 per cent of patients rheumatoid-like arthritis has appeared usually associated with abnormal hepatic function. Other abnormal manifestations have been fever, dermatitis, hematuria, albuminuria, anemia, leukopenia, lymphadenopathy, splenomegaly, and lupus cells have been found in blood preparations. These are considered to be late toxic manifestations, and actually may be suggestive of disseminated lupus erythematosis.

Drugs acting to interrupt somatic autonomic nerves are capable of producing a medical sympathectomy. Orally effective blocking agents are Hexamethonium, Pentolinium (Ansolysen), Chlorisondamine (Ecolid), and Mecamylamine (Inversine). The first three are quaternary ammonium drugs, and are absorbed to a varying and unpredictable extent from the gastro-intestinal tract. Inversine is an amine and is entirely absorbed from the gastro-intestinal tract. All four contain tetracovalent nitrogen (quaternary ammonium) as does acetylcholine. All have similar clinical effects and variations of action. There is no known metabolism for any of these, and all are excreted by the kidney. Increased effects of these drugs result from gastro-intestinal hypomotility which allows increased absorption; by renal failure which delays excretion; and by diminished intake of sodium. All of these blocking agents develop tolerance when they are used alone. The danger of postural hypotension is characteristic of all four, and ordinarily precludes lowering blood pressure to normal in any but erect

position. The autonomic blocking agents have the pharmacological effect of producing both sympatholysis and parasympatholysis. Sympatholysis produces desired antihypertensive effects, while parasympatholysis produces the undesired side-effects.

The major undesirable effects are retention of urine and intestinal paralysis. In the presence of impaired renal function these blocking drugs may accumulate, producing excessive drug action. Urinary retention can become an acute problem if these drugs are given in the presence of even incipient prostatic obstruction. When autonomic blocking agents are in use, constipation must be vigorously combatted as hypointestinal activity produces markedly increased blood levels of the drugs. Saline cathartics are required to control this type of constipation. Minor undesired effects include loss of visual accommodation, dryness of mucous membranes, diminution of normal sweating, and otitis media and parotitis have been occasionally observed from blocking antihypertensive drugs.

The correct dose of autonomic blocking drugs is unpredictable and must be determined individually. The oral effectiveness of the specific drugs is very different. Inversine is about fifty times as effective as Hexamethonium, ten times as effective as Ansolysen, and five times as effective as Ecolid.

When localized arteriosclerosis is present, violent fluctuation of blood pressure, which may be produced by drugs, may be very hazardous. Ischemia may be produced by a lower blood pressure in vessels that can not dilate as widely as the remainder of the vascular system, or by thrombosis beginning on an atherosclerotic plaque when blood flow is impaired. For these reasons cerebral thrombosis, mesenteric thrombosis, and coronary thrombosis have occurred, and must be kept in mind when arteriosclerotic patients are treated with antihypertensive drugs. Ischemia may also cause mental depression, temporary loss of memory, increased anginal pain and temporary diminution of renal function.

Hexamethonium has produced a fatal pulmonary disease characterized by fibrous pneumonitis in seriously ill patients with true malignant hypertension which has progressed to the point of azotemia. Hexamethonium is the only autonomic blocking agent associated with this complication, which has become less common in the last several years, probably because other ganglionic blocking drugs have become more popular, and because the control of hypertension has become

better with more experience. This curious complication has usually been associated with extreme tachypnea, and with respiratory rates of 60 per minute or more. These patients have not appeared orthopneic, but have actually been less dyspneic when lying flat. In the same small group of seriously ill true-malignant hypertensives in uremia, large doses of Inversine have produced a reversible nonfatal disturbance characterized by generalized and severe coarse tremors which disappear with sleep. Intense anxiety, sometimes hallucinations and a group of symptoms resembling delirium tremens rather than Parkinson's disease have occurred.

So far as the salt-steroid relationship is concerned, no drugs are available yet which will compete with salt-retaining hormones, and therefore sodium restriction is the only recourse. Too severe restriction stimulates salt-retaining hormone formation or may lead to low-salt syndrome, renal insufficiency and death. Therefore salt restriction to the degree necessary to lessen vascular reactions must be very carefully done in order to avoid both insufficient and excessive loss of sodium. There have been relatively few fatal reactions on this basis which indicates that the error is usually on the side of insufficient sodium depletion.

Hypertensive patients may develop a false sense of security simply because they are on antihypertensive treatment, although the treatment may be inadequate. Sudden withdrawal of antihypertensive drugs may result in rapid return of hypertension. If previously in the stage of malignant hypertension, that stage may return abruptly in a day or so after withdrawal of drugs with subsequent deterioration and death resulting from congestive heart failure, or from a cerebral vascular accident.

Hypotension may likewise be avoided, or the danger of hypotension be decreased by combining various drugs. Hydralazine (or Apresoline) alone is seldom given unless a surgical sympathectomy has been performed. A strict low sodium diet of .5 gm per day, if it can be obtained, may be as effective as that of hydralazine alone, and therefore may be substituted for hydralazine in combination with other drugs. The use of a combination of two or more powerful drugs in a single capsule or tablet may be hazardous. The dose of each drug must be adjusted to suit the individual. Side effects of ganglionic blocking drugs may be controlled by other blocking agents, such as Neostigmine (Prostigmin), or Bethanechol (Urecholine), which is a powerful parasympathetic stimulator.

Some of the side effects can be avoided by close observation when antihypertenisve treatment is instituted, by gradually increasing the dosage of antihypertensive drug or drugs, by combining different antihypertensive drugs, by having patients take their own blood pressure, and by warning patients of the possible side effects to be anticipated and avoided. Tolerance to the blocking drugs can usually be avoided or eliminated by the addition of Apresoline to the blocking agents. It is advisable and necessary to warn patients of the possibility of hypotension, episodes of weakness, light-headedness, or fainting and to sit or lie down immediately at the earliest symptoms.

Recent developments in the treatment of hypertension are rather remarkable. Lives have been prolonged. Cardiovascular-renal accidents and deterioration have been lessened. Regression in the basic hypertension has been demonstrated in numerous patients. However, the side effects of these drugs are not inconsiderable and the risk inherent in the administration of each of the hypotensive drugs is appreciable. Therefore, a significant diastolic hypertension should be present to justify the use of these powerful drugs. In future development of drugs of these types, it is to be hoped that the side effects may be minimized or that drugs may be developed to control the side effects.

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XIX

Norepinephrine in the Treatment of Shock

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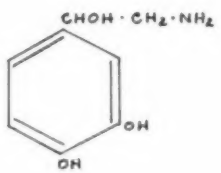
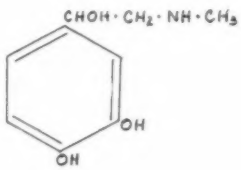
Norepinephrine is a hormone, closely related to epinephrine, and was first synthesized as early as 1904.[9] Norepinephrine has the following structural formula:



In this respect it differs from epinephrine only by the fact that the latter possesses one additional methyl group in the side chain:



Norepinephrine goes by various names: levophed, arterenol, noradrenaline. Norepinephrine occurs as two optical isomers: the laevorotatory form is pharmacologically a much more potent agent than the dextrorotatory form. The United States Pharmacopoea Standard epinephrine contains from ten to eighteen per cent of norepinephrine.[1]

Very little attention was given to this drug until 1946, when Von Euler[25-27] demonstrated its presence in mammalian tissues: spleen, heart, liver, adrenergic nerves. The adrenal tumors, pheochromocytomata, were found to be rich in norepinephrine.[7,11]

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Although closely associated with epinephrine in the natural state, and having a similar chemical structure, norepinephrine possesses interesting and significant differences from epinephrine in the pharmacological effect. Epinephrine acts as an over-all vasodilator and causes hypertenison by increasing cardiac output. Norepinephrine acts as an over-all vasoconstrictor without much change in cardiac output.[6]

The response to injection of epinephrine is hypertension, characterized by an increase in cardiac output, widening of pulse pressure, and an actual dilatation of small arterioles. The response to norepinephrine is much more like that seen in clinical hypertension, and is characterized by an increase in peripheral resistance (primarily due to increased small vessel tone) with no increase or an actual fall in cardiac output. Epinephrine causes an increase in the pulse rate. The systolic blood pressure rises, but the diastolic pressure remains unchanged. Norepinephrine, on the other hand, causes a marked peripheral vasoconstriction with a rise in both systolic and diastolic blood pressure. The metabolic effects of epinephrine (such as increased oxygen consumption, rise in body temperature and basal metabolic rate, hepatic glycogenolysis, rise in fasting blood sugar levels) are not seen after norepinephrine injection.[10]

An effective demonstration of the effect of norepinephrine was carried out by Goldenberg and associates.[8] Since the effect of norepinephrine on arterial pressure appears within a few seconds and subsides within three minutes after an intravenous infusion is stopped, these authors used a continuous intravenous flow. These authors studied the acute hypotension due to loss of vasomotor tone, which is encountered during and after thoracolumbar sympathectomy. It was found that the hypotension during the operation could be combatted successfully by the continuous administration of norepinephrine. They also reported successful restoration of blood pressure where the pressure had fallen to shock levels from massive hemorrhage. In these cases repeated transfusions of whole blood had failed to produce significant elevation of blood pressure. Likewise in three cases of severe hypotension associated with central vasomotor depression (shock from a prolonged operation, coma following an overdose of a barbiturate) norepinephrine proved successful in restoring and maintaining blood pressure.

Numerous other publications have attested to the value of norepinephrine in restoring and maintaining blood pressure in hemorrhage and traumatic shock.[15] In these conditions epinephrine is without

value or is actually harmful. DeLargy and his associates[4] showed that, when these two substances were infused in human muscular tissue, norepinephrine caused vasoconstriction of the intramuscular vascular bed, whereas epinephrine caused dilatation. Likewise, in cats, Burn and Hutcheon[3] found that infusion of epinephrine through the cat's hind leg, which had been denervated, caused a fall of blood pressure and dilatation of vessels in the denervated leg. On the other hand, infusion of norepinephrine produced a rise of blood pressure and constriction of vessels in the denervated leg.

According to Nathanson and Miller,[21] norepinephrine is of no value in the prevention or treatment of cardiac standstill. They chose for their study ten patients with complete heart block because the ventricular rate in heart block is relatively stable and is not influenced by extraneous factors, and because the sympathomimetic compounds are the only substances that effectively increase the activity of the ventricular pacemaker. Both norepinephrine and epinephrine were administered. A continual electrocardiogram was made and blood pressure recorded after thirty seconds and at one-minute intervals thereafter. The injection of norepinephrine caused an abrupt rise of blood pressure within thirty seconds, lasting five to eight minutes; however, the ventricular rate showed only a slight and transient increase. But after the administration of epinephrine, a pronounced and sustained increase was noted in the ventricular rate.

Several studies[17,24,28] have shown a consistent increase in coronary blood flow on intravenous injection of norepinephrine. The most striking increments (up to twenty times control value) occurred in animals with initial blood pressure at shock-level. There was actually a vasodilatation of coronary arteries.

The pulmonary circulation, when infused with norepinephrine, showed a rise in pulmonary arterial pressure, a rise in mean brachial arterial pressure, a decrease in cardiac output in about half of the experiments, a rise in pulmonary capillary pressure, and an increase in total peripheral resistance.[5] As to the renal circulation, Moyer, Skelton and Mills[20] showed that when norepinephrine is administered to normal dogs, it caused a marked reduction in renal blood flow, renal plasma flow, glomerular filtration rate, and maximum tubular transport of glucose. However, when used in shocked dogs in which renal function is markedly depressed, norepinephrine infusion actually increased renal function toward control levels. This was also observed in some patients who were anuric because of shock: following injection

of the drug, a significant increase of urinary output occurred. Sensenbach, Madison and Ochs[23] studied the effect of norepinephrine on the cerebral circulation of fifteen normotensive persons. The cerebral blood flow was measured before and during the height of pressor response induced by the intramuscular administration of "levophed in oil." They found that this was a powerful cerebral vasoconstrictor and that, although the cerebral blood flow was reduced, the oxygen consumption of the brain remained essentially unchanged. Norepinephrine has very slight stimulating effect on the central nervous system.[16] It has only a slight effect on sugar metabolism, its hyperglycemic action being far less pronounced than that of epinephrine.[13,14]

The acute toxicity of norepinephrine is somewhat less than that of epinephrine. Hoppe, Seffelin and Lands[12] established in mice LD_{50} values, showing that norepinephrine has a safety ratio approximately four times that of epinephrine. Harrison and co-editors state, in the section on shock of their textbook of internal medicine,[10] that when the use of this drug is prolonged, blood pressure may rise to alarming heights, acute pulmonary edema may occur, and ectopic ventricular rhythms may develop. The appearance of premature beats and of short bouts of ventricular tachycardia or irregularities call for immediate cessation of the administration of the drug.

There has been widespread use of norepinephrine in clinical medicine in the treatment of shock. If, in cases of shock, gross fluid losses have not occurred or if they have been replaced, there must be a considerable quantity of blood pooled in the venous system. There is evidence that this is the case. Thus, with the aid of radioactive isotopes, Nylin and Pannier[22] studied the mixing time of infused solutions with the circulation in various conditions and found that in shock it may be greatly prolonged. Whereas, ordinarily, a complete mixing occurs within three minutes, in shock, mixing may not be complete until after forty minutes. The implication is that blood is pooled in the vascular system in such a way that it does not partake in the circulation at the normal rate. Actual clinical experience has confirmed this view: if there is no fluid loss or if lost fluids have been replaced in cases of shock, the administration of norepinephrine is effective in restoring and maintaining blood pressure. Such conditions as the shock during or immediately after prolonged surgical procedures, especially thoracolumbar sympathectomies, during and after spinal anesthesia, during and immediately after resection of pheochromocytomata have been successfully treated with norepinephrine. This drug has proven effective in cases of shock-like states due to myocardial

infarction.[15,18,20] Shock-like states with peripheral vascular collapse complicating severe infections, such as pneumonia, meningitis (Waterhouse-Friderichsen syndrome) or overwhelming septicemia, have been successfully combatted with norepinephrine.[2]

Miller and his co-workers[19] reported good results in forty episodes of shock from various causes in 32 patients. There was no evidence in their cases that norepinephrine increased myocardial irritability. In no instance was heart failure precipitated or aggravated by the drug. It proved effective in shock following acute myocardial infarction, as interim therapy in oligemic shock until whole blood could be obtained, in shock following barbiturate poisoning, bulbar poliomyelitis, ruptured ectopic pregnancy, and anaphylactic shock.

An experience of our own with norepinephrine proved instructive. On September 16, 1955, the author was performing a laryngectomy on a 65 year old man at DePaul Hospital, St. Louis, Missouri. This patient's blood pressure prior to the operation was 140/80. The laryngectomy was being done under intravenous thiopentobarbital sodium anesthesia, supplemented with nitrous oxide, oxygen and ether. While the pharynx was being closed, near the end of the operation, cardiac arrest occurred. The chest was immediately opened and cardiac massage begun. The anesthetist began giving straight oxygen by controlled positive pressure through the endotracheal tube. Cardiac massage was continued for 50 minutes, and during this time solutions of calcium chloride, 10 ml (1.0 gm), and prostigmine, 3 ml (1.5 mg) were administered intracardially. After forty minutes heart beats commenced, at first feebly and intermittently, but soon becoming regular and rhythmical. We began to detect blood pressure readings of about 70/50. Norepinephrine was started intravenously at this point (4.0 ml in 1,000 ml of 5 per cent dextrose solution in distilled water) running in at approximately 20 drops per minute. This was continued for the next hour and a half. By this time the blood pressure was up to 140/80, and the patient had regained consciousness and had full use of his extremities. In the meantime we had completed the closure of the laryngectomy wound, had placed a water-sealed drain in the left pleural sac, and had closed the chest wall.

The blood pressure remained fairly stable for the next twelve hours, then began falling, for no apparent reason. When the pressure reached 92/66 we started intravenous norepinephrine again. During the next 24 hours the patient received 8 ml of the norepinephrine solution in 2,000 ml of 5 per cent dextrose solution in distilled water; this

was given at varying rates of flow, from eight drops per minute, maximum, down to zero. The blood pressure dropped to a minimum during this period of 90/60, and rose, on two occasions to a maximum of 150/68, but most of the time remained about 110/60. When the pressure rose the norepinephrine was discontinued, but the needle kept open and ready by having dextrose solution run in through another tube from a second flask. Finally the pressure stabilized around 150/60. During all this time the patient remained mentally alert, had good muscular function and good urinary output. The thoracotomy wound and the laryngectomy wound healed without difficulty, and there has been no apparent cerebral damage from the cardiac arrest. It was the opinion of all of us who worked with this patient that the norepinephrine administration was an effective means of raising and maintaining blood pressure in this shock-like state following cardiac arrest.

Norepinephrine is supplied in a sealed ampule, as a 0.2 per cent solution of the bitartrate (equivalent to a 0.1 per cent solution of the base) in 4 ml, and is to be administered by continuous intravenous drip, diluted in 1,000 ml of a 5 per cent dextrose solution in distilled water or 5 per cent dextrose solution in physiological saline solution, but not in saline solution alone nor in whole blood, because of the loss of potency due to oxidation of the drug. After allowing 2 or 3 ml of the solution to run into the vein per minute, the rate of flow is adjusted to maintain the desired blood pressure. Initially 20 to 40 drops per minute are used. Needless to say, the blood pressure must be checked every few minutes, as dangerously high levels of blood pressure may develop. When this is not possible, a single dose of 0.5 to 1.0 ml of a 1:1,000 solution of norepinephrine may be given intramuscularly, and repeated after two to six hours, as indicated by the clinical state and the blood pressure level.[9,10]

CONCLUSION

Norepinephrine is a hormone closely related to epinephrine, and shows a marked vasopressor effect. It is available for experimental and clinical use. This substance has proven useful in raising blood pressure, due to an over-all vasoconstrictor action. The various pharmacological properties of norepinephrine are discussed and evidence is reviewed to show its efficacy in the treatment of shock and shock-like states.

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XX

Anticoagulant Therapy

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Anticoagulant therapy is of interest to the otolaryngologist, not only when he is confronted with a bleeding emergency in a patient receiving such therapy, but also when he is confronted with intravenous clotting that formerly would have carried a fatal prognosis.

Successful treatment of cavernous sinus thrombosis with heparin combined with sulfanilamide derivatives was first reported by Lyons[1] and Schall[2] in 1941. In the same year Ershler and Blaisdell[3] reported one case similarly treated but complicated by massive hematuria. Wiesenfeld and Phillips[4] first reported a case successfully treated with heparin and penicillin. Reich, Likely, Yahr, and Baron[5] used dicumarol plus heparin and sulfadiazine. Other combinations of anticoagulants, sulfonamides, and antibiotics have subsequently been reported.

For a proper understanding of the indications and uses of anticoagulant therapy, a review of the blood clotting mechanism is necessary. This will be followed by a discussion of the clotting tests commonly used during anticoagulant therapy.

Blood clotting is initiated by contact of the blood with a foreign (rough or water-wettable) surface, whereby platelets rupture and liberate a lipoid factor called thromboplastinogenase. Thromboplastinogenase activates thromboplastinogen, which is present in the plasma and is also known as "antihemophilic globulin," to form thromboplastin. Whitby and Britton state that this formation of thromboplastinogen takes place in conjunction with calcium ions and two substances called factor VII and factor V. Thromboplastin is also liberated from injured tissues. Thromboplastin reacts with prothrombin in the presence of calcium ions and factor V to form thrombin. Prothrombin is present in the plasma. Its synthesis occurs in the liver and requires vitamin K. Thrombin reacts with fibrinogen, which is made in the liver and is found in the plasma, to form fibrin. The fibrinogen molecules are polymerized to form needle-shaped, crystal-like protofibrils which then become aligned into fiber strands by lateral associa-

tion. These fibrin needles have the capacity to contract. (Thrombin also accelerates the clotting mechanism by its labilizing action on platelets.) Intact platelets now adhere to the fibrin needles, forming large knots at their intersections and bending, twisting, and shortening the fibrin. Retraction of the clot therefore occurs, and thrombin is expressed from the clot. This clot retraction is known as syneresis.[6,7]

The platelets have three further important actions. They liberate a vasoconstrictor, they contain an accelerator of the prothrombin to thrombin mechanism, and they contain a substance which hastens the action of thrombin on fibrinogen.

The clotting tests mostly used by the otolaryngologist are 1) bleeding time, 2) coagulation or clotting time, 3) platelet count, 4) prothrombin time, and 5) clot retraction time. However, during anticoagulant therapy, the coagulation and prothrombin times are the ones usually determined.

The normal coagulation time as determined by the Lee-White method is 6 to 15 minutes. It is prolonged in a) hemophilia, in which there is a deficiency of thromboplastinogen, b) fibrinogen deficiency, c) severe prothrombin deficiency, and d) hyperheparinemia.

The prothrombin time requires a good laboratory where minute attention to detail is paid and where adequate controls are used. Quick's one-stage test is usually used. The result may be 10 to 25 seconds, but it is desirable to use thromboplastin of such potency as to give a normal time of about twelve and a half seconds. The prothrombin time is prolonged in vitamin K deficiency and in an excess of dicumarol.

The formation of an undesired, intravascular clot or thrombosis depends upon several predisposing factors. The first factor is a change of the vascular endothelium such as degeneration with atheroma or injury by accident or surgery. Another factor is slowing of the circulation. This may be due to shock or to lack of ambulation. A final factor is the agglutination of platelets. This is favored by the presence of foreign particles in the circulation, such as staphylococci.

Once a clot has formed, retraction takes place and thrombin is freed. If the blood circulation is slow, the freed thrombin is not washed away but begins its platelet labilizing action, thus starting further clot formation. This process may be repeated, giving growth to the thrombus.

The indications for anticoagulant therapy are many. Cardiac conditions include acute coronary occlusion, myocardial infarction, congestive heart failure, auricullar fibrillation, and mural heart thrombi. Each case must be evaluated on its own merits before a decision for anticoagulant therapy is to be made. Such therapy is used following vascular surgery. It may also be used, either prophylactically or therapeutically, for arterial embolization, frostbite, pulmonary embolization, acute thrombophlebitis, acute phlebothrombosis, after certain fractures, in certain post-traumatic cases, and in certain postoperative cases.

Anticoagulant therapy, along with chemotherapy and antibiotic therapy, has made possible the successful treatment of cavernous sinus thrombosis. It is also to be considered in treatment of lateral sinus thrombosis[8] and of thrombosis of other dural sinuses. Basilar artery thrombosis is also treated with anticoagulants and is of interest to the otolaryngolyogist because of the various cranial nerve manifestations of this condition.

Contraindications for anticoagulant therapy include the blood dyscrasias, hemorrhagic diathesis and tendencies, postoperative cases with poor hemostasis, brain and spinal cord surgery or recent injuries, active gastro-intestinal ulceration, malignant hypertension, bacterial endocarditis, and liver disease. Caution should be used in the presence of high fever and with drugs such as quinine and salicylates. These contraindications are especially for dicumarol and similar drugs with slow onset and long duration of action as compared with heparin which acts quickly and for a relatively short time.

For further discussion of the use of anticoagulant drugs, heparin will be considered first. It is a natural constituent of tissues. Commercial preparations are mostly of bovine origin. It acts by retarding the change of prothrombin to thrombin, by inactivating the thrombin that is already present, and by decreasing the agglutination of platelets.[9,10]

It acts quickly. One or two milligrams per kilo given intravenously will prolong the clotting time to four or five times normal within five to ten minutes. Normal clotting time is restored in two to four hours.

Several methods of administration may be used:

Intravenously, 50 mg may be given slowly every two to four hours. The clotting time should be determined before each dose until the proper dosage is established. Continuous intravenous drip with 200 mg in 1000 cc of saline or 5 per cent glucose may be given. The suggested rate of 20 drops per minute is varied depending upon the effect upon the clotting time.

Intramuscularly, heparin may be given 50 mg every three to four hours or, in a concentrated form of five or ten per cent solution, 100 mg every six to eight hours. This latter form may also be used subcutaneously. The addition of procaine and hyaluronidase to the injection avoids considerable pain.

Depo-solution heparin, N.N.R., containing 200 mg per cc in gelatin and dextrose is given intramuscularly in doses of 300 to 400 mg and is said to be effective for as long as 24 hours. The injected muscle should not be massaged since massaging will hasten absorption. Repository forms with a vasoconstrictor are also available but should be avoided in coronary disease. Sublingual wafers of 125 mg of heparin sodium are said to be absorbed in ten minutes, therapeutic action being obtained in 30 minutes and maintained for four hours.

The optimum dosage of heparin should increase the clotting time to two or three times the normal value.

Heparin is mainly excreted through the kidneys, but small amounts are destroyed by heparinase.

If bleeding occurs from overdosage, the dose or frequency may be decreased. Transfusion with whole fresh blood is used in an emergency. Protamine sulfate is a specific antidote. It has an antithromboplastin action itself but has a greater affinity for heparin; 50 mg given intravenously will usually reduce the clotting time to therapeutic levels within a few minutes. However, 150 mg can be given intravenously in saline over a period of one hour. Toluidine blue also counteracts heparin. It should be given slowly intravenously in a dose of 4 to 6 mg per kilo. Too rapid administration may lead to nausea and vomiting.

The other most commonly used anticoagulant is bishydroxycoumarin (Dicumarol). It is similar in its structure to vitamin K and exerts a competitive inhibition of the synthesis of prothrombin. It also decreases the agglutinability and adhesiveness of the platelets. It

is used by mouth; 300 mg are given at first, followed by 200 mg the second day, 100 or 200 mg the third day, and 50 to 100 mg a day thereafter.

The prothrombin time should be performed daily until the dosage is regulated, then less often until weekly determinations suffice. A prothrombin time of 22 to 35 seconds is desired with a control of about 15 seconds.

Ethyl biscoumacetate (Tromexan) is similar to dicumarol but is quick acting. It is also more expensive than dicumarol. It is given 1.5 gm at once or in doses divided over a period of 24 hours, then 0.6 to 0.9 gm a day. Wright[11] suggests that it be given 1.2 to 1.5 gm along with 200 to 300 mg of dicumarol the first day. The patient may then be carried on the Dicumarol alone.

Cyclocumarol (Cumapyran) is given 0.1 to 0.2 gm the first day, then 25 to 50 mg a day. Other coumarin derivatives are 3-(1-phenylpropyl)-4 hydroxycoumarin (Marcumar) and warfarin sodium.

If bleeding occurs, Dicumarol or its derivatives should be stopped immediately. If hemorrhage is severe, fresh whole blood transfusions are given. Vitamin K_1 (Phytonadione) works the most quickly of all the vitamin K preparations. Its action starts to take place in a half hour. 10 to 20 mg are given orally every three to six hours as needed. Wright advises that long term patients keep a small supply on hand and take 10 mg if bleeding should occur, then call the doctor. An intravenous dose of 100 to 150 mg will return the prothrombin time to safe levels in approximately six hours. Parenteral vitamin K_1 oxide, 1 gm, and repeated doses of menadione sodium bisulfite (Hykinone) may be used but require several hours before action takes place.

Indanedione has a different chemical structure from Dicumarol and Tromexan, but is counteracted by vitamin K_1.

Paritol and Treburon are considered as too toxic for use.

Hirudin is the anticoagulant formed by the leech. It interferes with the enzymatic action of thrombin on fibrinogen. It has no use in modern medicine.

Phosphorylated hesperidin and Plasmin are other anticoagulants that have not gained accepted usage as yet.

SUMMARY

Anticoagulants, along with the sulfonamides and antibiotics, have made successful treatment of cavernous sinus thrombosis and basilar artery thrombosis possible. They are used, both therapeutically and prophylactically, for a variety of medical and surgical conditions. They are to be considered in the management of lateral sinus thrombosis. The prompt availability of a good laboratory is essential to the effective and safe administration of anticoagulants. Heparin and Dicumarol are the most widely used anticoagulant agents.

FRISCO BUILDING

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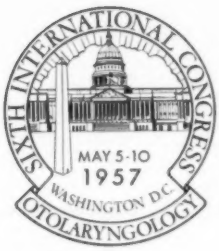

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

PROGRAM

SUNDAY, MAY 5, 1957

8:30 a.m.-4:00 p.m.

Registration, Presidential Ballroom, Hotel Statler
Scientific and Technical Exhibits open
Hospitality Lounge for Foreign Members open
Ladies' Committee Hospitality Lounge open

5:00 p.m.

Convocation, Constitution Hall

Congress to be Opened by the Honorable RICHARD M. NIXON, Vice-President of the United States

6:00 p.m.

President's Reception and Buffet Supper, Pan American Union (Informal)

MONDAY, MAY 6

8:30 a.m.-5:00 p.m.

Registration, West Checkroom, Hotel Statler — *Open each day*
Exhibits open — *during*
Hospitality Lounges open — *these hours*

9:00 a.m.-12:00 noon

Plenary Session, Presidential Ballroom, Hotel Statler

CHRONIC SUPPURATION OF THE TEMPORAL BONE

Openers: Dr. Marcus Diamant, Halmstad, Sweden
Anatomical and Etiological Factors in Chronic Middle Ear Disease

Prof. Luzius Rüedi, Zürich, Switzerland
Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone

Prof. Horst Wullstein, Würzburg, Germany
Surgical Repair for Improvement of Hearing in Chronic Otitis Media

Discussers: Mr. A. Tumarkin, F.R.C.S., Liverpool, England
Prof. Juan Manuel Tato, Buenos Aires, Arg'ntina
Mr. T. E. Cawthorne, F.R.C.S., London, Eng.
Prof. Fritz Zöllner, Freiburg, Germany

Sightseeing tours in and about Washington (available daily)

2:00-5:00 p.m.

General Sessions, 5 simultaneous sections (rooms to be designated)
Film Session, Presidential Ballroom
Sightseeing tours (available daily)
Medical tours to Walter Reed Medical Center
Armed Forces Institute of Pathology
Navy Medical Center, Bethesda, Maryland
National Institutes of Health, Bethesda
Forest Glen Speech and Hearing Center

(These tours available each afternoon)

8:00 p.m.

Reception and tour, National Gallery of Art

TUESDAY, MAY 7

9:00 a.m.-12:00 noon

General Sessions, 5 simultaneous sections
Film Session, Presidential Ballroom
Special all-day program for ladies including tour and luncheon

2:00-5:00 p.m.

General Sessions, 5 simultaneous sections
Film Session, Presidential Ballroom
Special ladies' program continued during afternoon

8:30 p.m.

Concert by the National Symphony and the Howard University Choir, Constitution Hall
Program of musical Americana

WEDNESDAY, MAY 8

9:00-9:30 a.m.

Special address, Presidential Ballroom

DOCTORS AS DIPLOMATS

Howard Rusk, M.D., Director, Institute of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, New York

9:30 a.m.-12:30 p.m.

Plenary Session, Presidential Ballroom

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT

Openers: PROF. HANS SELYE, Montreal, Canada
Introduction

PROF. MICHELE ARSLAN, Padua, Italy
The Upper Respiratory Tract

DR. LESLIE GAY, Baltimore, Maryland
The Lower Respiratory Tract

Discussers: Sir Victor Negus, London, England
Prof. Branimir Gusic, Zagreb, Yugoslavia
Dr. Aubrey G. Rawlins, San Francisco, California, U.S.A.
Dr. Henry L. Williams, Rochester, Minnesota, U.S.A.

2:00-5:00 p.m.

General Sessions, 5 simultaneous sections
Film Session, Presidential Ballroom

5:00-6:00 p.m.

Business meeting, International Committee (Official Delegates)

6:00-7:00 p.m.

Dinner for Official Delegates and wives, South American Room (formal)

THURSDAY, MAY 9

9:00 a.m.-12:00 noon

General Sessions, 5 simultaneous sections
Film Session, Presidential Ballroom

2:00-5:00 p.m.

General Sessions, 5 simultaneous sections
Film Session, Presidential Ballroom

7:00-8:00 p.m.

Congress cocktail party, Sheraton-Carlton Hotel

8:15 p.m.

Grand Banquet, Presidential Ballroom and Congressional Room, Statler Hotel (formal)

FRIDAY, MAY 10

9:00 a.m.-12:00 noon

Plenary Session, Presidential Ballroom

PAPILLOMA OF THE LARYNX

Openers: Prof. Jo Ono, Tokyo, Japan
Etiology

Prof. Plinio de Mattos Barretto, Sao Paulo, Brazil
Diagnosis

Mr. F.C.W. Capps, London, England
Therapy

Discussers: Prof. C. A. Hamberger, Göteberg, Sweden
Dr. Pedro Hernandez Gonzalo, Havana, Cuba
Prof. Eelco Huizinga, Groningen, Netherlands
Prof. Albert von Riccabona, Vienna, Austria

2:00-5:00 p.m.

General Sessions, 5 simultaneous sections
Film Session, Presidential Ballroom

The following activities will be scheduled every day:

8:30-5:00

Registration
Scientific Exhibits
Technical Exhibits
Hospitality Lounge for Foreign Members
Ladies' Committee Hospitality Lounge

Morning and afternoon
Hours to be announced
Sightseeing tours

Each afternoon
Hours to be announced
Medical tours

PAPERS OF THE GENERAL SECTIONS

Die Prognose der operativen Hörbesserung bei chronischer Mittelohrentzündung	Zöllner, Fritz Freiburg, Germany	*German*
The accessory air spaces of the middle ear: morphology, physiology and pathology	Tumarkin, Ivan Alexis Liverpool, England	*English*
Ten years of tympanoplasty in chronic suppurative otitis	DePrest, R. A. Bruges, Belgium	*English*
Tympanoplastic surgery and its long term results	Goto, Shuji Nagoya-Shi, Japan	*English*
Modifizierte zweizeitige tympanoplastik bei chronischer otitis media	Brunar, Max Graz, Austria	*German*
Surgical aspects of tympanoplasty	Verhoeven, Louis Antwerp, Belgium	*English*
Chronic suppuration of the ear and functional surgery	Fabbi, Fabio Bologna, Italy	*English*
Principles of hearing preservation in surgery for chronic ear disease	Juers, A. L. Louisville, Ky.	*English*
Reconstruction of sound conduction of the middle ear	Pick, Emery I. Los Angeles, Calif.	*English*
Zur ätiologie des Mittelohr cholesteatoms	Link, Rud. Berlin-Charlottenburg, Germany	*German*
A new concept of the pathogenesis of aural cholesteatoma based upon studies of the early development of the middle ear	Guggenheim, Paul Topeka, Kansas	*English*
Remarks on the x-ray appearance of the mastoid process in health and suppurative disease	Alexander, A. B. London, England	*English*

Considerations sur la tomographie du temporal	Agazzi, Carlo Cova, P. L., and Senaldi, M. Milan, Italy	*French*
Ueber das sogenannte primäre Cholesteatom des äusseren Gehörganges	Hörbst, Ludwig Innsbruck, Austria	*German*
Perilabyrinthitis	Cawthorne, T. E. London, England	*English*
Sobre microcirugia funcional en las otorreas	Garcia-Ibanez, L. Valencia, Spain	*Spanish*
Tratamiento de las otitis media cronicas supuradas con lavaje permanente con hialuronidasa	Rellan, Raul Maria Buenos Aires, Argentina	*Spanish*
Les fistules labyrinthiques dan les otites moyennes suppurées chroniques	Lapouge, Jean Nice, France	*French*
Radical surgery on the temporal bone and the labyrinth for the improvement of hearing	Niho, Shijo Yokohama, Japan	*English*
Clinical significance of the new conception of otitis media	Nishihata, T., and Nishihata, H. Tokyo, Japan	*English*
The problem of the sterile otitis media	Siirala, Urpo Turku, Finland	*English*
Modern surgery for chronic suppurative otitis media and mastoiditis	Morrison, W. W. New York, N. Y.	*English*
Pathology of otitis media	Friedmann, I. London, England	*English*

The Schwartze mastoidectomy: its value at the present time	McKenzie, William London, England	*English*
La chirurgie fonctionelle des fenêtres labyrinthiques	Portmann, Michel Bordeaux, France	*French*
The first direct prelabyrinthine petrotomy and extended tympano-mastoidectomy with preservation of labyrinthine function	Precechtel, Antonin Prague, Czechoslovakia	*English*
Past and present treatment of otogenous labyrinthitis	Miodonski, Jan Krakow, Poland	*English*
The effect of head movement on positional nystagmus	Aschan, G., Bergstedt, M., Drettner, B., Nylen, C. O., and Stahle, J. Stockholm, Sweden	*English*
The electric goniometer test. A new vestibular function test	Honjo, Shoichi Ube-shi, Japan	*English*
A contribution to clinical electronystagmography	Piroda, E. Cagliari, Italy	*English*
Improved equipment for vestibular testing	Guillemin, Victor, Jr. and Torok, Nicholas Chicago, Illinois	*English*
Nystagmus problems in brain tumor	Morimoto, Masamori Niigata City, Japan	*English*
Farmacodinamia de los drogas inhibidoras del sistema vestibular	Tato, J. M., Fernandez, C., and Rius, M. Buenos Aires, Argentina	*Spanish*

Anomalies in the recorded movement of the eyes during rotatory optokinetic and caloric stimulation in normal subjects	Omerod, F. C. London, England	*English*
Temperature transmission in the caloric test in the os temporal under various anatomical and pathological conditions	Jako, G., Fleischmann, L., and Missurat, T. Budapest, Hungary	*English*
Nouvelle aiguille a paracentése	Bors, C. Bucharest, Rumania	*French*
Fenestración modificado y movilización del estribo (estapedioclasia). Tecnicas, resultados y juicio critico	Antoli-Candela Cebrian, F. Madrid, Spain	*Spanish*
Mobilisation de l'étrier: échecs et espoirs	Clerc, Pierre Paris, France	*French*
La mobilisation de l'étrier dans l'otosclérose	Arslanian, Nubar Genoa, Italy	*French*
Stapedolysis in the treatment of otosclerosis	Goodhill, Victor Los Angeles, Calif.	*English*
Four years' experience with mobilization of the stapes	Werth, Rudolph Tel Hashomer, Israel	*English*
Modified Rosen's operation	Pruvot, Maurice Lille, France	*English*
Mobilisation de l'étrier et ouverture de la fenêtre ovale	Guillon, Henri Paris, France	*French*
Nuestra experienca con la operación de Rosen	Bertran Escanaverino, Raul E. and Basterrechea, Claudio, Jr. Havana, Cuba	*Spanish*

Stapes mobilization and fenestration of oval window for otosclerotic deafness	Rosen, Samuel New York, N. Y.	*English*
The mechanism of hearing loss in early cases of labyrinthine hydrops	Tonndorf, Juergen Iowa City, Iowa	*English*
The humoral cochlear circulation	Borghesan, Ettore Palermo, Italy	*English*
Structural changes in the inner ear under the influence of sound	Engstrom, Hans Göteborg, Sweden	*English*
Sudden perceptive deafness	van Dishoeck, H.A.E., and Bierman, Th. A. Leyden, Netherlands	*English*
The normal hearing reference zero	Glorig, Aram Los Angeles, Calif.	*English*
The present state of the treatment of the hard of hearing in Japan	Kashiwado, Teiichi Tokyo, Japan	*English*
Congenital deafness: analysis of pathological findings	Kelemen, George Boston, Mass.	*English*
On recruitment testing	Palva, Tauno Turku, Finland	*English*
Loudness measurement in a mobile decompression chamber	Pothoven, W. J. Groningen, Netherlands	*English*
The first and second branchial arches: their developmental history and adult contribution to the auditory apparatus in man	Anson, B. J. Chicago, Illinois and Bast, T. H. Madison, Wisconsin	*English*

Prevention of adverse effects of streptomycin on the ear	Ozaki, Tomoharu Nara-ken, Japan	*English*
Research in normal hearing threshold	Albrite, J. P. and Shutts, R. Edwin Washington, D.C.	*English*
Sodium bicarbonate injection in the prevention of motion sickness	Muta, Minoru Osaka, Japan	*English*
New investigations on seasickness	Nieuwenhuijsen, J. H. Rotterdam, Netherlands	*English*
Nistagmus pervertido. Su significacion clinica	Riesco-MacClure, J. S., and Martinez, Alfonso Santiago, Chile	*Spanish*
Psychogenic vertigo: the importance of its recognition	Moore, Burness E. and Atkinson, Miles New York, N. Y.	*English*
Electrophysiological and cytochemical studies on ototoxicity of dehydrostreptomycin	Nakamura, Fumio Kioto, Japan	*English*
Ueber Spätfolgen der petrogenen Hirnabsesse	Fleischmann, L. Budapest, Hungary	*German*
Chemodectoma or tumor of the glomus jugulare of tympanic body	Capps, F.C.W. London, England	*English*
The function testing of inner ear windows	Horiguti, Sinsaki Tokyo, Japan	*English*
Intravascular agglutination of the blood. A factor in several diseases and disorders of the ear	Fowler, Edmund P. New York, N. Y.	*English*

An oto-audiological investigation of premature children	Campanelli, P. A. Henner, R., and Pollock, F. J. Chicago, Illinois	*English*
An objective test method for the evaluation of middle ear function	Holcomb, A. L. Los Angeles, California	*English*
Hearing conservation program in U. S. Air Force	Kraus, Ralph N. Randolph Field, Texas	*English*
Studies on the clinical masking level	Harbert, Fred Philadelphia, Pa.	*English*
Progression of cochlear nerve degeneration in otosclerosis as estimated by audiogram	Roy, S.R.D.N. Tamluk, India	*English*
Anatomical and clinical reclassification of the tympanic spaces and the eustachian tube	Schwarzbart, Adolph A. Tel-Aviv, Israel	*English*
Stapes mobilization: lasting results of 1 and 2 years	Muerman, Y. and Meurman, O. Helsinki, Finland	*English*
Improvement of hearing in otosclerosis by means of stapes mobilization	Ronis, B. J., and Myers, David Philadelphia, Pa.	*English*
Visualization of the fenestra ovalis in the operation for mobilization of the stapes	Bellucci, Richard J. New York, N. Y.	*English*
Mobilization of the stapes in otosclerotic deafness	Alexander, Lucian W. New Orleans, La.	*English*
Progress and observations of transtympanic mobilization of the stapes	Scheer, Alan A. New York, N. Y.	*English*

Considerations on mobilization of the stapes for otosclerosis	Myerson, M. C. Beverly Hills, California	*English*
Ausmeisselung der Steigbügel Grundplatte	Heerman, Hans Essen, Germany	*German*
Heparin in the treatment of Meniere's disease	Schenck, Harry P. Philadelphia, Pa.	*English*
Ablation therapy for Meniere's disease	Schuknecht, Harold F. Detroit, Michigan	*English*
Simultaneous registration of vestibular nystagmus and counter-rolling of the eyes	Kuilman, J. The Hague, Netherlands	*English*
Occupational hearing loss	Fox, Meyer S. Milwaukee, Wisconsin	*English*
Evaluation of the signs of end-organ deafness	Simonton, K. M. Rochester, Minn.	*English*
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Normal and abnormal activity of the human otoliths	Sullivan, J. A. and Johnson, Walter Toronto, Canada	*English*
Le traitement chirurgical des vertiges par l'ouverture du sac endolymphatique	Portmann, Georges Bordeaux, France	*French*
Les otites adhesives et leur traitement chirurgical	Maspetiol, Roger Paris, France	*French*

L'ostéite condensante de l'oreille moyenne	Andrieu-Guitrancourt, S. A., and Laumonier, R. Rouen, France	*French*
Le tympanelectronique endaural	Aubert, Maurice, and Miquelis, Eugene Nice, France	*French*
Ueber den Einfluss des Labyrinthreizes auf die Funktion des autonomen Nervensystems	Fujisaki, Shigemi Osaka, Japan	*German*
The effect of succinylocholine on the middle ear muscles and on transmission of sound through the middle ear	Gisselsson, L. Lund, Sweden	*English*
Retroauriculaire fistule symptom	Grec, Stamatios Petalas Budapest, Hungary	*French*
Valeur de la conservation du tissu adipeux pieleve sur le vivant et sur le cadavre dans la chirurgie de l'os temporal	Penha, Guilherme Coimbra, Portugal	*French*
Surdites centrales avec recruitment dans le syndrome de Wallenberg	Greiner, G. F., Theibaut, F., and Mengus, M. Strasbourg, France	*French*
Audiocirugia. Recuperación de las sorderas por otorrea or secuelas	Antoni-Candela Piquer, F. Valencia, Spain	*Spanish*
Chronic middle ear infection among Filipinos	Garcia, Tierry F. and Fernando, Asuncion Manila, Philippines	*English*

Topic diagnosis in facial nerve paresis	Kristensen, Harold K. Copenhagen, Denmark	*English*
Reflexions au sugjet de abcès encéphaliques oto-rhinogènes	Larroude, Carlos Lisbon, Portugal	*French*
The dry fenestration technique	Lempert, Julius New York, N. Y.	*English*
The fenestration operation: lasting results through refinements in technique	Farrior, J. Brown Tampa, Florida	*English*
Non-chromaffin paraganglioma of the head	Lederer, F. L., Skolnik, E. M., Fornatto, E. J., and Soboroff, B. J. Chicago, Illinois	*English*
Sur le colèsthéatome du rocher du temporal et de la base du crane	Racoveanu, Virgil & Anghelide, Radu Bucharest, Rumania	*French*
Myringoplasty: Plastic repair of ear drum perforations	Pietrantoni, Luigi and Bocca, Ettore Milan, Italy	*English*
External umbo blebs. Non-inflammatory lipoid vesicles of the tympanic membrane	Armstrong, B. W. Charlotte, N. C.	*English*
Chloromycetin en las otitis cronicas supuradas	Tombolini, Jorge J. San Lorenzo, Argentina	*Spanish*
The labyrinth test in the syndrome of Barre	Demetriades, Theodore D. Athens, Greece	*English*

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Surgical treatment of chronic sinusitis with special reference to the external frontal approach	Kurosu, Minokichi Tokyo, Japan	*English*
Transnasal destruction of the pituitary gland for secondary carcinomatosis in hormone dependent neoplasms	Bateman, G. H. London, England	*English*
Recherches histo-pathologique sur l'agmidale dans les maladies collagenes	Ferreri, Giorgi Rome, Italy	*English*
The pterygopalatine ganglion and its anesthesia applied to otalgic, glossopharyngeal pains and certain forms of vasomotor disturbances of the nose	Laskiewicz, A. London, England	*English*
Vascular surgery in otolaryngology	Loré, John M. New York, N. Y.	*English*
Experiences in the treatment of the allergic nasal polyp by the injection of hydrocortisone T.B.A. and Prednisolone T.B.A.	Myers, David and Ronis, B. J. Philadelphia, Pa.	*English*
El colageno en la fisiopathologia del escleroma	Sanchez Cortes, Jose, Maranjo, Ramon, and Montes de Oca, Edmundo Guadalajara, Mexico	*Spanish*
Malignant granuloma of the nose	Ellis, Maxwell P. London, England	*English*
Clinical study of local treatment of allergic rhinitis	Evans, W. H. Youngstown, Ohio	*English*

Versuche postoperative mesenchymale Reaktionen zu beeinflüssen	Malecki, J. Lodz, Poland	*German*
Vasomotor rhinitis	Goldman, Joseph L. New York, N. Y.	*English*
Nasal allergy	Aiyar, R. D. Denbigh, Wales	*English*
Stress and hypadrenocorticism: the implications in otolaryngology	Goldman, Herbert B. Rockville Center, New York, and Tintera, John W. Yonkers, N. Y.	*English*
Optimum dosage therapy in respiratory allergy and in infection	Hansel, French K.	*English*
Allergie u. respirationstrakt	Hussarek, Max Vienna, Austria	*German*
The treatment of rhinoscleroma locally by aureomycin	El-Mofty, Aly Cairo, Egypt	*English*
Rhinitis hipertroficas en Venezuela	Gonzalez Vera, P. Caracas, Venezuela	*Spanish*
The effect of antrotomies with negative findings on the improvement of nutritional disturbances in infant age	Baraat Budapest, Hungary	*English*
The congenital malformation of nasal structure	Horowitz, Michael S. London, England	*English*
The surgical treatment of nasal polypi	Korkis, F. Boyes London, England	*English*

Radical frontal sinus surgery	Goodale, Robert L. Boston, Mass.	*English*
Pathology of the sphenoid	Hirsch, Oscar Boston, Mass.	*English*
Branchial anomalies	Albers, G. Donald Grand Rapids, Mich.	*English*
Choanal polyps	Van Alyea, O. E. Chicago, Illinois	*English*
Chronic osteitis of the maxilla in relation to maxillary sinusitis	Fleming, W. E. Melbourne, Australia	*English*
Transethmoid-sphenoidal hypophysectomy in metastasis of breast cancer	Escher, Franz Bern, Switzerland	*English*
Rhinosporidiosis in nasopharynx	Burad, Ermin Ankara, Turkey	*English*
Observations on the exchange of fluid in the nose and respiratory tract	Negus, Victor London, England	*English*
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Rhinoplasty in Japan with special reference to second grade of physiologic saddle nose	Ishii, Toshi Yokohama, Japan	*English*
New things in plastic surgery of the nose	Seltzer, Albert P. Philadelphia, Pa.	*English*
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Anatomy and function of the nasal vestibule	Williams, Russell I. Cheyenne, Wyo.	*English*
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A biochemical and cytological study of tonsils	Saito, Hideo, and Nakamura, Shiro Tokyo, Japan	*English*
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Surgical access to the nasopharynx	Wilson, C. P. London, England	*English*
Malignant tumors of the paranasal sinuses	Struben, W. H. Amsterdam, Netherlands	*English*
Spätresultaten in der behandlung der operativen verlätzung des gesichtsnerven im säuglingsalter	Kallay, Franz Budapest, Hungary	*German*
Results in cases of rhinolalia aperta	Missura, T. Budapest, Hungary	*English*
New developments in plastic surgery of the nose and ear	Converse, John M. New York, N. Y.	*English*

Submucous fibrosis of the palate	DeSa, Joseph V. Bombay, India	*English*
Elongated styloid process: symptoms and treatment	Eagle, Watt W. Durham, N. C.	*English*
Ectopic salivary adenomas	Harrison, Kenneth Manchester, England	*English*
Mixed tumors of the palate	Montreuil, Fernand Montreal,Canada	*English*
End results of the management of parotid tumor	Perzik, S. L. Beverly Hills, California	*English*
Nasopharyngeal fibroma	Satyanarayana, C. Madras, India	*English*
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Anesthesia in peroral endoscopy: a new method	Adler, H. J., Reed, W. A., and Frerichs, D. A. Phoenix, Arizona	*English*
Neurogenic tumors of upper respiratory tract	Ash, James E. Bethesda, Md.	*English*
Submucous fibrosis of oropharynx (a collagen disorder)	Rao, R. Venkata New Delhi, India	*English*
Adenoidectomy under visualization	Holt, John A. B. Charleston, W. Va.	*English*
Sinography especially with reference to block dissection of the neck	Frenckner, Paul Stockholm, Sweden	*English*

Tracheotomy	Steinberg, Edgar I. Los Angeles, California	*English*
The innervation of the larynx	Rethi, A. Budapest, Hungary	*English*
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Magnetic removal of foreign bodies from the stomach and duodenum	Equen, Murdock Atlanta, Ga.	*English*
The laryngologist and resuscitation of the newborn	Goff, Willard F. Seattle, Wash.	*English*
Discrimination entre les diverses thérapeutiques des tumeurs malignes de l'amygdale palatine	Cambrelin, G. Brussels, Belgium	*French*
Composite grafts for the repair of alar defects	Becker, Oscar J. Chicago, Illinois	*English*
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Le rôle de l'air dans la vibration des cordes vocales	Vallancien, Bernard Paris, France	*French*
The significance of endobronchial lesions in the management of pulmonary tuberculosis	O'Keefe, John J. Philadelphia, Pa.	*English*

Les oesophagites par reflux	Gaillard, J., and Mounier-Kuhn, P. Lyon, France	*French*
Observation upon the experimental use of radioactive isotopes within the larynx	Pressman, Joel J., Dowdy, Andrew, Libby, Raymond, and Fields, Max Los Angeles, California	*English*
Vocal rehabilitation of paralytic dysphonia	Arnold, Godfrey E. New York, N. Y.	*English*
Vocal cord polyps: psychiatric observation on the personality structure of patients with habitual dysphonia	Heaver, Lynwood New York, N. Y.	*English*
Stenosing peptic esophagitis	Friedberg, Stanton A. Chicago, Illinois	*English*
Tratamiento de la ventilación pulmonar insuficiente en enfermos sin obstrucción laringea	Barani, Julio C. Montevideo, Uruguay	*Spanish*
Quiste intralaringea	Revuelta Alonso, Rene, and Mateo Oma, Luis Havana, Cuba	*Spanish*
Papiloma de laringe	Ameriso, Jose Rosario, Argentina	*Spanish*
Considérations sur a papilomatose du larynx	da Costa Quinta, Antonio Lisbon, Portugal	*French*

Papilloma of the larynx: a case of protracted duration, 1878-1955	Jesberg, Norman Los Angeles, California	*English*
Surgical treatment of multiple recurrent obstructive papillomata of the larynx	Woodman, De Graaf New York, N. Y.	*English*
Ueber das pH in der O.R.L. insbesondere bei Papillome des larynx	Yannoulis, G. E. Thessaloniki, Greece	*German*
Laringofisura en el tratamiento de la Papilomatosis laringea	Celis Perez, A. Valencia, Venezuela	*Spanish*
La papillome et son problème thérapeutique	de Sanson, Raul D. Rio de Janeiro, Brazil	*French*
Reconstrucción quirurigica de laringes estenosadas	Aleman Caballero, Rene Seville, Spain	*Spanish*
Carcinoma of the larynx	Jackson, Chevalier L. Philadelphia, Pa.	*English*
The classification of carcinoma of the larynx based on topography	King, G. David Boston, Mass.	*English*
Some selected problems in cancer of the larynx	Orton, Henry B. Newark, N. J.	*English*
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Traitement du cancer de la base de la langue	Labayle, J., and Huet, R. D. Paris, France	*French*

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Datos sobre casuistica y terapeutica del cancer laryngea	Ager Muguerza, Enrique Madrid, Spain	*Spanish*
Consideraciones sobre etiologica, caracteristicas, localización y distribución del cancer de la laringe en Espana. Derivada de nuestra estadistica	Paredero del Bosque, J. Madrid, Spain	*Spanish*
Preventive neck dissection in cancer of the larynx	Putney, F. Johnson Philadelphia, Pa.	*English*
Laryngo-esophagectomy: primary closure with laryngo-tracheal autograft	Som, Max J. New York, N. Y.	*English*
Etude critique des nouveaux procédés d'explanation bronchoesophagienne	Soulas, Andre, Paris, France, and Mounier-Kuhn, P. Lyon, France	*French*
Observations on the voice and their application	Lodge, William O. Halifax, England	*English*
Neuere Methoden der allgemeinen Betaübung für die bronchoscopie	Riecker, O. E. Wuppertal-Barmen, Germany	*German*
Tissue culture studies in juvenile papilloma of the larynx	Brewer, David W., and Kalter, Seymour Syracuse, N. Y.	*English*

Traitement des papillomes larynges des enfants par les antibiotiques	Zakrezewski, Alexandre, and Sobocynski, Andre Posnan, Poland	*French*
Consideration etiologique à-propos d'un cas de tracheomalacia	Mounier-Kuhn, P., Heimindinger, E., and Klotz, G. Strasbourg, France	*French*
Considerations sur le papillomatose du larynx	Negrea, A., and Bors, D. Bucharest, Rumania	*French*
Some considerations about papilloma of the larynx	Amarante, Rubem C. L. Rio de Janeiro, Brazil	*English*
Treatment of serious esophagus stenosis	Koleszar, G. Budapest, Hungary	*English*
Treatment of laryngeal papilloma in children with resin of podophyllin	DeVido, G., and Megighan, D. Treviso, Italy	*English*
Postlaryngectomy pharynx fistula and the ribbon muscles	Singer, Rudolf New York, N. Y.	*English*
Contact ulcer: new observations and therapy	von Leden, Hans and Moore, Paul Chicago, Illinois	*English*
(Incomplete)	Groen, J. J. Utrecht, Netherlands	*English*

(Incomplete)	Aubin, Andre Paris, France	*French*
(Incomplete)	Melampi, Mario Genoa, Italy	*French*
Case of pharyngolith	Ulug Resat, Ali Ankara, Turkey	*English*
Repair of tympanic membrane perforations	Elia, Joseph C. West Haven, Conn.	*English*
Surgical management of complete and incomplete atresia of the posterior nares	Beinfeld, Henry H. Brooklyn, N. Y.	*English*
Sinusitis and non-specific endogenous ocular inflammations	Lubart, Joseph New York, N. Y.	*English*
Stress rhinology	Poos, Edgar E. Detroit, Mich.	*English*
Psychological preparation of children for surgery	Coleman, Lester L. New York, N. Y.	*English*
The culmination phenomenon and frequency pattern of thermic nystagmus	Torok, Nicholas Chicago, Ill.	*English*
Nose and nasality	Froeschels, Emil New York, N. Y.	*English*
A new method of decompression in malignant exophthalmos	Biber, J. J. Delaware, Ohio	*English*
Case report: Sudden fatal termination in case of otitis media	Loomis, George Winona, Minn.	*English*
An analysis of blood and vascular factors in the prophylaxis of tonsillo-adenoidal surgery	Coyle, James E. Detroit, Mich.	*English*

Conservative management of chronic otitis media	Sanderson, Bruce A. San Diego, Calif.	*English*
Bronchogenic carcinoma, with report of a case	Strong, M. Stuart, and Sommers, Sheldon C. Boston, Mass.	*English*
The role of the vocalis muscle in human phonation	Fink, R. Bernard New York, N. Y.	*English*
The adenoid-tonsil problem: a review of 5000 cases	Rigg, James P. Grand Junction, Colorado	*English*
Bilateral thyrotomy: revaluation after 5 years	Kemler, Joseph I. Baltimore, Md.	*English*
Hypopharyngeal diverticulum	Shepard, Willis B. Eugene, Oregon	*English*
Bleeding problems and the otolaryngologist	Simmons, Marvin W. Fresno, Calif.	*English*
Postoperative hemorrhage related to atmospheric changes	Utrata, Joseph Chillicothe, Ohio	*English*
Epistaxis. The need for new and improved methods of treatment	Woodruff, George H. Joliet, Illinois	*English*
Transantral decompression for malignant exophthalmos	Walker, James S. and Porter, G. LeRoy Urbana, Illinois	*English*
A short study of papilloma of the larynx	Shanks, Joseph Chicago, Illinois	*English*
Physiology of breathing and colds	Josephson, Emanuel M. New York, N. Y.	*English*

Ultrasonics, resonance and deafness	Angeluscheff, Zhivo D. New York, N. Y.	*English*
Quelques aspects particuliers en ce qui concerne les modifications auditives consecutives au traitement chirurgical des suppurations oto-mastoidiennes	Neuman, H., Falutz, S., Muntenescu, M., and Craciun, E. Bucharest, Rumania	*French*
The problem of the papillomatous larynx as a precancerous condition	Marin, Aurel, and Mestes, Eugen Bucharest, Rumania	*English*
Recherches experimentales dans l'allergie nasale	Laurian, N., Lengyel, A., Mestes, E., Ursu, E., Cociumian, L., Bujum, O., and Obreja, S. Bucharest, Rumania	*French*
Contributions a la conduite therapeutique de la papillomatose laryngee	Nerescu, V. Bucharest, Rumania	*French*
The allergic tissue with special reference to eosinophils and ground substance alteration	Hlavacek, Vladimir Prague, Czechoslovakia	*English*

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Applications for the presentation of films have been sufficient to provide 18 hours of motion pictures which will run simultaneously with the presentation of scientific papers.

THE RESPONSIBILITIES OF THE MEDICAL PROFESSION IN THE USE OF X-RAYS AND OTHER IONIZING RADIATION

STATEMENT BY THE UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION

The United Nations General Assembly, being aware of the problems in public health that are created by the developments of atomic energy, established a Scientific Committee on the Effects of Atomic Radiation. This Committee has considered that one of its most urgent tasks was to collect as much information as possible on the amount of radiation to which man is exposed today, and on the effects of this radiation. Since it has become evident that radiation due to diagnostic radiology and to radiotherapy constitutes a substantial proportion of the total radiation received by the human race, the Committee considers it desirable to draw attention to information that has been obtained on this subject.

Modern medicine has contributed to the control of many diseases and has substantially prolonged the span of human life. These results have depended in part on the use of radiation in the detection, diagnosis and treatment of disease. There are, however, few examples of scientific progress that are not attended by some disadvantages, however slight. It is desirable therefore to review objectively the possible present or future consequences of increased irradiation of populations which result from these medical applications of radiation.

GENERAL SURVEY OF THE IRRADIATION OF HUMAN BEINGS

Man has always been exposed to some irradiation from natural sources. To this has now been added, as a result of modern discoveries and the applications of ionizing radiation and radioactivity, certain forms of artificial irradiation.

Natural irradiation is due to: 1) cosmic radiation: 2) "background" gamma radiation from radioactive substances present locally in the earth, rock or building materials, and from disintegration prod-

ucts of radon in air; 3) radiations emitted from natural radioelements such as potassium 40, radium, radon and carbon 14 which are incorporated in the body.

The amount of this natural radiation varies with locality, but has been estimated as usually delivering between 70 and 170 mrems per year to the gonads. Of this total, the major contributions are of about 45 per cent from local gamma radiations, 30 per cent from cosmic rays and 20 per cent from body potassium 40.[1]

Artificial irradiation is derived from: 1) the contamination of the environment, the atmosphere, or water by radioactive waste from atomic industries or from users of radioelements; 2) the radioactive fallout, at greater or lesser distances from the source, or radioactivity resulting from the explosion of nuclear devices; 3) the occupational exposure of certain groups of workers: medical practitioners, radiologists, dentists, nurses, atomic energy workers, uranium or thorium miners, and the industrial or scientific users of radiation generators or radioactive isotopes; 4) the medical use of x-rays, other ionizing radiations and radioelements in the detection, diagnosis, investigation and treatment of human diseases; 5) the use of certain devices which emit radiation, such as television receivers, watches with luminous dials, and the x-rays generators used for the purpose of fitting shoes.

The amount of artificial radiation must vary considerably in different countries and we have inadequate information as to the overall significance of these factors. In certain countries where estimates have been made, it appears that the greatest gonad irradiation of the population is due to diagnostic radiological procedures, the amount from this source about equalling that from all natural sources in certain instances. The total present contribution from occupational exposure, from the products of atomic industries from radiotherapy and from the radiating devices mentioned above is likely to be very considerably smaller. That from radioactive fallout to the gonads appears at present to be in the region of 1 per cent of the natural gonad irradiation in most areas.[2]

Both the magnitude and the significance of these various sources are under review by the Committee. Since medical irradiation accounts for a substantial if not the major proportion of all artificial exposure, it is important that its magnitude should be known accurately for different countries and circumstances. The possibility of making such an assessment depends upon the help of the medical pro-

fession, and particularly on the adequacy and availability of records kept by doctors, dentists and organizations responsible for the use of ionizing radiation.

RADIATION HAZARDS

The medical use of radiation is clearly of the utmost value in the prevention, diagnosis, investigation and treatment of human disease, but the possible effects of this irradiation of individuals require examination.

Generally speaking, the irradiation of living beings may produce radiobiological effects either on the irradiated individual himself or, through him, on his descendants; the former being termed somatic and the latter genetic effects. Somatic effects vary according to the different organs or tissues affected, and range from slight and reversible disturbances such as cutaneous erythema to the induction of leukaemia or of other malignant diseases. The possible reversibility of the somatic effects of radiation received in small doses at low dose rates encourages the belief that there are permissible doses of radiation which will not cause completely irreversible or significant somatic damage. The threshold for occasional somatic damage may, however, prove to be low. In the case of genetic effects, on the other hand, there may be no threshold. These effects increase with a frequency corresponding to the total amount of radiation received by the germinal tissues, and in the great majority of cases, are adverse.

Many other factors complicate the interpretation of radiobiological effects. The differences between whole and partial body radiation, between a single exposure and continuous irradiation, or between the effects of different types of radiation are still imperfectly understood. Biological differences in the radiosensitivity of various tissues, or of the tissues of people of different age or sex, obviously influence the nature of radiation hazards. It is clear, however, that any radiation of gonads, and any substantial irradiation of other tissues, involve a chance of significant damage which requires assessment.

GENERAL RECOMMENDATIONS REGARDING THE MEDICAL AND OCCUPATIONAL IRRADIATION OF HUMAN BEINGS

The radiological profession, through the International Commission on Radiological Protection,[3] has undertaken a valuable and responsible duty in defining maximum permissible limits of exposure for the main radiation hazards.

The establishment of these maximum permissible levels for those who are occupationally exposed to radiation depends on the view that there are doses which, in the light of our present knowledge, do not cause detectable somatic injury in the individual irradiated; and on the consideration that the number of individuals concerned is small enough for the genetic effects on the whole population to be negligible. For the gonads, or for irradiation of the whole body, the levels are such as to exclude doses greater than 0.3 rem in any week or 3.0 rem in any 13 weeks, or a sustained irradiation rate greater than 5 rem per year. These values imply that no total dose of over 50 rem will have been received by the gonads by the age of 30, or of over 200 rem by the whole body by the age of 60, in any occupationally exposed person.[4]

As regards irradiation of the whole population, it is considered prudent to limit the average dose to germinal tissues from artificial sources to the order of magnitude of that received from all natural sources.

In considering the extent to which the population is irradiated for medical purposes, it is essentially the genetic hazard which is involved although it seems possible that in certain circumstances somatic injury may occur occasionally after low doses of radiation arise. Otherwise, the relevant dose is that indicating the mean gonad irradiation among the population as a whole up to the end of the average reproductive period.

The extent of such genetic irradiation from diagnostic procedures has been found to be equal to at least 100 per cent of all natural radiation in two countries,[5] and that from a third equalled at least 22 per cent of this figure.[3] Even before obtaining more exact values for these and other countries, it is clear that the exposure can be substantial in countries with extensive medical facilities, and that it is essential to consider any ways in which this exposure could be reduced without detriment to the existing or developing value of medical radiology.

The Committee is therefore anxious to obtain the help of radiologists in suggesting through appropriate governmental channels any methods by which this total exposure could be reduced and in estimating the amount of reduction that might be expected from any such methods. In particular it would be valuable to know how much the radiation to the gonads could be reduced a) by improved design or shielding of equipment; b) by fuller training of any individuals using radiographic or fluorscopic equipment; c) by any local shielding of the gonads that is practicable, especially during abdominal or pelvic

examination; d) by the use of techniques involving radiography rather than fluorscopy when full information can be obtained by this means; e) by improvement of administrative arrangements designed to obviate unnecessary repetition of identical examinations of the same subject; f) by a general study of certain medical conditions such as that of peptic ulcers, to identify the circumstances in which the establishment of a radiological diagnosis has or has not a definite influence upon the treatment or prognosis given.

SUMMARY

The Scientific Committee on the Effects of Atomic Radiation established by the United Nations General Assembly accepts the view that the irradiation of human beings, and especially of their germinal tissue, has certain undesirable effects.

Information received so far indicates that, in certain countries (Sweden, United Kingdom, United States of America), by far the most important artificial source of such irradiation is the use of radiological methods of diagnosis and that this may be equal in importance to radiation from all natural sources. It is possible that such radiation may be having a significant genetic effect on the population as a whole.

The Committee is fully aware of the importance and value of the medical use of radiations but wishes to draw the attention of the medical profession to these facts and to the need for a more accurate estimate of the amount of exposure from this source. The help of the medical profession would be most valuable to make it possible to obtain fuller information on this subject.

The Committee would be particularly grateful for information through appropriate governmental channels on ways in which the medical irradiation of the population can be reduced without diminishing the true value of radiology in diagnosis or treatment.

REFERENCES

1. Reports sent by India, Sweden, the United Kingdom and the United States of America.

2. Reports sent by the United Kingdom and the United States of America.

3. Report of the International Commission on Radiological Protection, published in the *British Journal of Radiology*, Supplement 6, of December 1954, in the *Journal Francais d'electro-radiologie*, No. 10, of October 1955, etc.

4. Report of the International Commission on Radiological Protection, published in the *British Journal of Radiology*, Supplement 6, of December 1954, in the *Journal Francais d'electro-radiologie*, No. 10, of October 1955, etc.

5. Sweden, United States of America.

6. United Kingdom.

Abstracts of Current Articles

EAR

Audiological Investigations in Noise Deafness.

Boenninghaus, H. G., Roser, D., and Rossberg, G.: Ztschr. f. Laryng. Rhinol. Otol. 34:614-625 (Sept.) 1955.

The authors tried to assess the problem of noise at its source in a locomotive boiler factory and made a very thorough audiological and clinical examination of the ears of 14 noise deafened workers. No early cases were included in this study.

The structure of the buildings visited and the character of the jobs and working conditions of the boiler makers were described. Noise measurements were first charted at the factory. It was found that pneumatic pressure hammers made the highest and most disagreeable noise.

Noises under different working conditions were recorded in phons and decibels. Special instruments and gauges were employed. The noises were almost equal in their characteristics to typical "white" noise. The ages of the workers, employed from 3 to 39 years, were between 35 and 62. Some of them had more or less inner ear deafness and most, except the older ones, used ear plugs; only a few used cotton. Special consideration was given to audiological investigations at the clinic.

Speech, and hearing acuity, including speech audiometry, were compared and related to the age of the workers, the nature and duration of the noise.

A striking observation was made: with short duration and smaller hearing losses, a ganglion type was found, but after longer duration of work, the hair cell type of noise damage was noted.

The presence of the recruitment phenomena from damage to the organ of Corti as a result of long-acting noises was so striking that the authors could not accept the report of a ganglion type of hearing loss under such conditions. In all cases of positive recruitment they

found a striking loss of discrimination. They feel that catarrhal conditions of the eustachian tube aggravate inner ear noise deafness and believe that it is not possible to get an exact evaluation of the quality and quantity of noise deafness without several methods of ear examinations, and finding the amount of discrimination loss.

GLICK

Sudden Occurrence of Unilateral Deafness.

Schroeder, H. O.: Ztschr. f. Laryng. Rhinol. Otol. 34:630-639 (Sept.) 1955.

This report represents the results of a study on 16 patients with unilateral more or less sudden inner ear deafness to evaluate some causative factors in labyrinth hydrops without vertigo, and some other symptoms. These patients were found to have an irregular vegetative disturbance of the inner ear believed to be caused by cervical irritations of the sympathetic nervous system. Structural abnormalities of the spine were evaluated by x-ray examinations. The therapeutic measures followed were based on the work of Moritz reported before the Austrian Otologic Society in 1952.

The author outlines the characteristic cervical findings in sudden inner ear disturbances as described by Moritz. He is still of the opinion that noninflammatory diseases of the inner ear are as described by Ménière in 1861.

Since the publications of Hallpike and Cairns in 1938 most authorities have accepted the histologic explanation for labyrinth hydrops as the causative factor in Ménière's disease. The author's observations support the validity of Moritz' "Cervical Sympathetic Irritations Syndrome" as a causative factor.

GLICK

Clinical Determination of Abnormal Auditory Adaptation.

Carhart, Raymond: A.M.A. Arch. Otolaryng. 65:32-39 (Jan.) 1957.

The author has studied the "relapse" phenomenon described by Hallpike and Hood. By this is meant the decay of a tone after an initial response. Normally, a tone 5 db above threshold can be heard for 60 seconds. In certain abnormal conditions, especially where re-

cruitment is present, the tone is heard over a shorter period of time. In the studies by the author, the tone intensity was increased in 5 db steps until a point was reached where the patient heard it for a full 60 seconds, or until 60 db above the patient's threshold was reached. This decay phenomenon occurs in Ménière's syndrome and in acoustic trauma and does not occur in presbycusis. It is a phenomenon similar to recruitment and can be used in the same way. A "threshold tone decay test" was developed but the author states that the patterns have not yet been well worked out and that some unexplainable, astonishing curves occur, for which there is as yet no explanation. Carhart thinks the "relapse" phenomenon is sufficiently independent from the presently measured auditory reactions so that further elucidation of the phenomenon can probably lead to classification of sensorineural involvements into new clinical subgroups. This test promises to be a useful one.

HILDING

Objective Audiometry. Utilization of an Unconditioned Muscle Reflex for the Determination of Sound Perception.

Kobrak, H. G.: A.M.A. Arch. Otolaryng. 65:26-31 (Jan.) 1957.

The author has demonstrated both in experimental animals and in man that the muscle reflex of the stapedius and tensor tympani muscles can be used as an objective test of cochlear response to sound. The stapedius muscle responds better than the tensor tympani. The cat, dog and rabbit he found were better to test than the guinea pig.

Certain physiological conditions are necessary:

1. The test must be made under local anesthesia, or extremely light general anesthesia.

2. Exposure of the middle ear must be done without damage to the structures of the neck, or ear.

3. The middle ear must be kept free of blood.

4. The onset of the stimulus must be immediate, i.e., "rectangular," in order to avoid the "creeping in" effect.

5. The proper experimental animal must be used.

6. The reflex is best observed in young animals.

In man, direct observation is possible only if there is a perforation through the drum and the tendon of the stapedius muscle is visible and there is essentially no disease in the middle ear. Kobrak, however, developed a test in which the ear with the intact drum may be tested. He fixed a tiny mirror to the umbo by means of small droplets of ointment and then made recordings of the excursion of the light reflected off the mirror during sound stimulation. He found that there were, in general, two movements: the vibratory movement due to the sound waves and a retraction movement due to the contraction of the ear muscles. He measured the angle of the excursion of the reflected ray of light and from this calculated the linear movement of the center of the drum. These studies are patterned somewhat after those of Dr. W. Köhler of 50 years ago. Since this test is still only feasible in an otological laboratory, the author is working on a simpler method which will record the magnified eardrum rotations outside the body.

HILDING

NOSE

Meticorten (Prednisone) and Meticortelone (Predisolone) in the Treatment of Allergic Disorders.

Arbesman, C. E., and Ebrenreich, R. J.: Jour. of Allergy 27:297-304 (July) 1956.

The authors used these two new steroids in the management of a variety of allergic disorders including cases of asthma, perennial and seasonal allergic rhinitis, nasal polyps, atopic eczema, chronic urticaria, contact dermatitis and gastrointestinal allergy and found them as helpful as their precursors, cortisone and hydrocortisone, but with less of the side effects of the older preparations.

In all, 101 patients were treated, some of them having multiple allergic manifestations. All had complete diagnostic work-ups and had received the usual therapeutic measures to which they had failed to respond adequately. Forty-two of the 101 patients had been taking either ACTH, cortisone, hydrocortisone or all three, and some for as long as four years.

The dosage with the new steroids is initially 5 mg four times daily for seven days. The amount is gradually lowered each week until a minimum maintenance dosage is established or the drug discon-

tinued. The minimum daily dosage requirement is usually between 7½ and 10 mg.

There is no apparent difference in the effectiveness and side effects of the two preparations, meticorten, and meticortelone, but milligram for milligram they are three to five times more potent than the older steroids. They are to be preferred also because they do not produce sodium retention and potassium depletion with a consequent lessening of the side effects.

All preparations, however, are similar in that the symptoms return with the discontinuance of the drug. The authors caution that the newer preparations even though more potent and less severe in side effects than the others are not a substitute for the standard methods of therapy and should not be prescribed promiscuously but reserved for those cases in which the other measures are ineffective.

VAN ALYEA

Treatment of Seasonal and Perennial Allergic Rhinitis with Prednisone and Prednisolone.

Brown, E. B., and Scideman, T.: Jour. of Allergy 27:305-311 (July) 1956.

Each year since 1952 the authors have attempted to relieve the symptoms of hay fever with first, cortisone, then hydrocortisone with equally unsatisfactory results. In the 1955 season, however, the new steroids, prednisone (meticorten) and prednisolone (meticortelone) were tried on 157 patients and good to excellent results were obtained in most cases. In a control group of 78 patients only 19 received 75 per cent relief by other means of therapy.

In another study six patients with severe perennial allergic rhinitis were treated between March and November 1955. Other treatment had failed in previous years, but with small doses (15-20 mg) daily of the steroids these patients were kept relatively free of symptoms over long periods. When the drugs were withdrawn relief continued for 14 to 42 days and when reinstituted a smaller dose sufficed. Side effects were noted but not of a severity to require discontinuance of the drug. In each case nasal symptoms recurred when the dosage was reduced below 7.5 mg per day.

VAN ALYEA

LARYNX

The Effects of Hormone Disturbances on the Larynx.

Leicher, H., and Matzker, J.: Ztschr. f. Laryngol. Rhinol. Otol. 34:569-578 (Sept.) 1955.

In this paper the authors call attention to the fact that no publication has appeared in the German literature on the relationship between internal secretions and diseases of the ear and the upper air and food passages since a monograph by Leicher in 1928.

They state that the development and the function of the larynx stand in close relationship with the glands of internal secretion. Also, some disorders of the sex glands and other structures concerned with internal secretions have been known for a long time to cause functional disturbances in voice production. Contributions to this subject include observations on involvement of the nasal mucosa, the gingiva and the larynx from thyroid, pituitary, and adrenal dysfunctions. The authors describe the symptoms, voice changes, and laryngeal findings in cases of laryngopathia gravidarum, myxedema, acromegaly, and laryngospasm in tetany.

In a case of laryngopathia gravidarum in a mother and daughter seen for the first time, a reciprocal relationship existing between fetus and mother, and the possibility of a hereditary tendency to laryngopathia gravidarum was considered. In a case of idopathic tetany in an adult, the importance of the calcium-ion in the blood was indicated. Relief of laryngospasm and bronchotetany by intravenous injection of 10 per cent calcium chloride was shown.

GLICK

MISCELLANEOUS

Total Unilateral Pulmonary Collapse.

Lubert, M., and Krause, G. R.: Radiol. 67:2:175-185 (Aug.) 1956.

This is a complete analysis of the lateral roentgenogram in unilateral pulmonary collapse. Serial roentgenograms were taken of the chest in a number of cases. As a second approach roentgenologic principles inherent in the silhouette sign as described by Felson were applied.

Appearance of the lateral roentgenogram is discussed. Diagrams, schematic representations and 24 chest roentgenograms illustrate findings described in the text. Changes as seen in unilateral collapse of the upper, middle, lower lobe and entire lung are depicted.

JORSTAD

Anomalous Course of Left Pulmonary Artery with Respiratory Obstruction.

Wittenborg, Martin H., Tantiwongse, Thavi, and Rosenberg, Barbara F.: Radiol. 67:3:339-345 (Sept.) 1956.

Anatomic variations of intrathoracic vessels, particularly of the aortic arch are discussed briefly as a cause of respiratory obstruction in infants and children. The authors report two cases in detail in which the left pulmonary artery follows an anomalous course producing definite respiratory obstruction. The condition is amenable to surgical correction. They cite three additional cases recently published.

Spot films during fluoroscopy show the constant finding of indentation of the esophagus. The mechanics of this finding is illustrated by diagrams.

An eleven week old infant with symptoms since birth failed to respond to general and supportive therapy. At post mortem several associated vascular anomalies were found. The second case with an identical pulmonary artery deformity died at the age of three weeks. Autopsy also revealed a coarction of the aorta.

Summary of findings in these two and three additional cases seen during the past two years are tabulated. Six chest radiographs show essential changes. The embryologic explanation of the anomalous course of the left pulmonary artery is discussed. Clinical signs are an expiratory wheezing beginning at or shortly after birth with spells of cyanosis and convulsions.

All the patients were males. The successful surgical correction in the one case (Potts) is added incentive to pursue specific diagnosis and encourage vigorous therapy. Five references are included.

JORSTAD

Roentgen Changes Following Radical Neck Dissection.

Simpson, S. Aaron, Gordon, Sewell S., Jorgens, Joseph, and Rigler, Les G.: Radiol. 67:5:704-713 (Nov.) 1956.

Chest roentgenograms of 31 unilateral and 2 bilateral neck dissections were evaluated from the standpoint of characteristic roentgen findings. Routine posteroanterior chest roentgenograms were made preoperative and following the operative procedure.

The side operated upon presents the following salient anatomical features, important from a radiographic standpoint: general soft tissue deficiency, absence of sternomastoid muscle, frequent excision of XI cranial nerve with innervation of the trapezius muscle, subperiosteal calcification of upper border of clavicle, possible injury to the phrenic nerve with partial or complete paralysis of the diaphragm.

In tabulating these changes manifested on the roentgenograms and considering two of them as the lower limit upon which to base a suspicion of abnormality, the authors deem it possible to suggest the diagnosis of radical neck dissection from the roentgenogram alone in approximately 80 per cent of the cases. Sixteen roentgenograms, one diagram, and two references are included.

JORSTAD

Mediastinal Carinal Bronchogenic Cysts.

Davis, James G., and Simonton, John H.: Radiol. 67:3:391-395 (Sept.) 1956.

The embryology, pathology, clinical findings, and differential diagnosis of mediastinal carinal bronchogenic cysts of congenital origin is discussed.

Clinical course depends upon the location and the presence or absence of infection. Many of the cysts produce no symptoms. Cough is fairly common. Substernal pain may occur.

Three case reports are given. Eight radiographs show characteristic mediastinal changes. Eight references are included.

JORSTAD

Canker Sores from Allergy to Weak Organic Acids (Citric and Acetic). Case Report and Clinical Study.

Tuft, Louis, and Ettelson, L. N.: J. Allergy 27:6:536-543 (Nov.) 1956.

Tuft and Ettelson report the case of a male patient, aged 37, treated for nasal allergy, asthma, and migraine who since the age of five had been subject to frequent outbreaks of oral canker sores. Clinical investigation indicated that the latter usually followed the ingestion of foods, drugs, or carbonated beverages containing citric acid. Direct application of citric acid crystals to the oral mucosa repeatedly reproduced the ulcer in the patient, but not in the controls. Mucous membrane contact tests with substances containing citric acid gave similar positive results; likewise, tests with other weak organic acids contained in foods gave positive reactions to some of these, especially to acetic acid. Avoidance of foods containing the positive reactors was followed by marked relief not only of the ulcers but also of the general "toxic" symptoms previously regarded as functional.

VAN ALYEA

The Use of Phenothiazine Derivatives in Ear, Nose, and Throat Surgery.

Reinke, C.: Ztschr. f. Laryng. Rhinol. Otol. 34:501-510 (Aug.) 1955.

This article deals chiefly with the author's experiences with some of the newer tranquillizing and depressant drugs derived from phenothiazine. He evaluates their sedative or stress diminishing effects which may lessen the dose of narcotics and anesthetic agents needed in preoperative and postoperative cases. He mentions the underlying principle that has been advanced for their use and offers an explanation for the complex action of these drugs on the autonomic nervous system.

The drugs derived from phenothiazine carried trade names such as Phenergan Atosil, Largactil or Megaphen, which were particularly known as such in Germany, and had been used in 380 operations under local and general anesthesia. Among other members of the phenothiazine group employed were Diparcol Latibon and Dolantiin. We recognize Dolantin in this country as Meperidine or Demerol, and Largactil as Chlorpromazine or Thorazine, which is chemically related to the antihistamines. Each drug possesses a significant pharmacological pattern of response or degree of narcosis when administerd in three types of anesthesia levels. These levels were referred to as "light

anesthesia," "potentiated narcosis" and "artificial hibernation." With the latter method, the author has had little or no experience. He feels that the introduction of these sedatives before, during, and after surgery, has resulted in a decided change in anesthesiology and predicts that the otolaryngologist may some day be his own anesthetist.

He presents indications and contraindications for their use, suggestions for their administration and dosage, and the treatment of some undesired side reactions.

GLICK

On the Unusual Manifestations of Pemphigus Vulgaris in Otolaryngology.

Schnurbusch, F.: Ztschr. f. Laryng. Rhinol. Otol. 34:520-524 (Aug.) 1955.

The author brings out some important points about the origin, distribution and behavior of pemphigus vulgaris, a recurring skin disease which also affects mucous membranes. The important characteristic is the formation of vesicles. The lesions develop both in the acute and chronic forms and vary in size and distribution. In at least half of the cases the lesions first start in the oral cavities, and a number of patients come to the otolaryngologist. Diagnosis may be difficult depending upon the history, clinical picture, method of vesicle formation, localization, healing, and scar formation. The author mentions other diseases in differential diagnosis. He states that at the present time a poor prognosis is recognized. The author describes two patients observed who had unusual manifestations which he considered of special interest to the otolaryngologist. These were involvement of the esophagus with extensive cicatricial stenosis; affection of the ear drum with subsequent atrophic scarring; involvement of the nose and accessory sinus cavities with atresia of the vestibule on one side and stenosis on the other side. He explained the relationship that may exist between hyaluronic acid and hyaluronidase and one may assume, from the therapeutic effect with the application of cortisone and echinacine, a disturbance in balance between mucopolysaccharides and mucopolysaccharoses.

GLICK

Notices

AN INTERNATIONAL CONFERENCE ON AUDIOLOGY

1. There will be an International Conference on Audiology in St. Louis beginning the afternoon of May 13 and continuing through May 16, 1957. The Conference will be included as part of Tours 2 and 3 (to the Middle West and Far West) that are being planned in connection with the Sixth International Congress of Otolaryngology to be held in Washington in May 1957.

2. The following themes will be discussed: a) The Assessment of Auditory Function; b) The Physiology of Audition; c) The Relation of Hearing Loss to Noise Exposure.

The program has not been finally crystallized but it is likely that the majority of papers will be invited ones. One afternoon has been set aside for a small number of contributed papers relevant to the themes of the Conference. Persons desiring to contribute papers should submit an abstract to the Program Chairman no later than March 1, 1957. The aim of the Conference is to have scientists and clinical workers share views and findings on the above subjects. Visits to local audiological and otological facilities will be included.

3. The Chase Hotel is the official hotel of the Conference, but housing elsewhere at varying prices will be available. There will be a nominal registration fee, not yet determined.

4. The Conference is being partly supported by a grant from the National Science Foundation to the Central Institute for the Deaf.

5. The Program Chairman is Dr. Ira J. Hirsh, Research Associate, Central Institute for the Deaf, 818 South Kingshighway, St. Louis 10, Missouri.

6. The National Organizing Committee is:

Raymond Carhart, Ph.D., Professor of Audiology, Northwestern University

Hallowell Davis, M.D., Director of Research, Central Institute for the Deaf; Research Professor of Otolaryngology and Professor of Physiology, Washington University School of Medicine

Harvey Fletcher, Ph.D., Director of Research, Brigham Young University; formerly Physical Research Director, Bell Telephone Laboratories

William G. Hardy, Ph.D., Director, Speech and Hearing Center; Associate Professor of Otolaryngology and Environmental Medicine, Johns Hopkins Hospital, Baltimore, Maryland.

John Lindsay, M.D., Professor of Otolaryngology, University of Chicago

Philip E. Meltzer, M.D., Professor of Otolaryngology, Tufts University Medical School

S. S. Stevens, Ph.D., Professor of Psychology, Harvard University

S. R. Silverman, Ph.D., Director of Central Institute for the Deaf; Professor of Audiology, Washington University; Chairman

7. Additional plans will be communicated as they are formulated. For information write to: S. R. Silverman, Chairman, International Conference on Audiology, Central Institute for the Deaf, 818 South Kingshighway, St. Louis 10, Missouri.

INTERNATIONAL VOICE CONFERENCE

Some forty distingushed laryngologists, physiologists, physicists, and voice scientists from outstanding research centers in the United States, Europe and Asia are sending delegates to appear on the program of the International Voice Cenference in Chicago May 20-22, 1957.

The various days will be devoted to the following subjects: Monday—Research on Physiology of Voice Production; Tuesday—Clinical Procedures in Diagnosis and Training; Wednesday—Relation of Hearing to Voice.

Participation in the Conference will be by prior registration. Any further information may be obtained from Dr. Hans von Leden, 30 North Michigan Ave., Chicago 2, Ill.

ACADEMY HOME STUDY COURSES

The 1956-1957 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

AMERICAN LARYNGOLOGICAL ASSOCIATION

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to:

Dr. Edwin N. Broyles,
Editor Transactions
1100 North Charles St.
Baltimore 1, Md.

UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 30 to October 6, 1957. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

UNIVERSITY OF ILLINOIS

The next Laryngology and Bronchoesophagology course to be given by the University of Illinois College of Medicine is scheduled for the period November 4 to 16, 1957. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

TULANE UNIVERSITY OF LOUISIANA SCHOOL OF MEDICINE

The three year residency in otolaryngology offered at Charity Hospital of Louisiana at New Orleans on the Tulane University of Louisiana School of Medicine service is designed to qualify the holder for the examinations of the American Board of Otolaryngology and the practice of all phases of otolaryngology and endscopy.

Candidates must be graduates of a class A medical school and must have completed a minimum of one year of general internship. An additional year of residency in internal medicine or general surgery is desirable but not essential.

All work is under the direct supervision of members of the Tulane Department of Otolaryngology, who are also members of the Charity Hospital Otolaryngological Staff; they are available at all times for instruction and guidance. Basic sciences are offered throughout each year of the residency during the academic year. The resident also participates in the program of the Speech and Hearing Center at the Tulane University School of Medicine.

The hospital year extends from July 1 of one year to June 30 of the following year.

Applications should be addressed to the Chairman of the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, 1430 Tulane Ave., New Orleans 12, Louisiana.

TEMPLE UNIVERSITY
SCHOOL OF MEDICINE AND HOSPITAL

Postgraduate Course in Bronchoesophagology, May 20-31, 1957; September 9-20, 1957.

Postgraduate Course in Laryngology and Laryngeal Surgery, November 4-15, 1957.

These courses are all to be given in the Department of Laryngology and Broncho-Esophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris. The tuition fee for each course is $250.00. Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pa.

MOUNT SINAI HOSPITAL

A special course in Rhinoplasty and Reconstructive Surgery of the Septum will be given at Mount Sinai Hospital, May 13 - 18, 1957, after the Sixth International Congress. It will be under the direction of Dr. Irving B. Goldman, and will be limited to foreign doctors, on a full scholarship basis.

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 13 - 27, 1957, also by Dr. Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University.

Candidates for either course should apply to the Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

GERMAN SOCIETY OF THROAT, NOSE AND EAR SPECIALISTS

The German Society of Throat, Nose and Ear Specialists (Deutsche Gesellschaft der Hals-Nasen-Ohrenärzte) will hold its 28th annual meeting June 3-6, 1957, in Baden-Baden under the chairmanship of Professor Doctor Hünermann.

The program: Prof. Dr. F. Zöllner, Freiburg i. Br., "Hörverbessernde Operationen bei entzündlich bedingten Mittelohrveränderungen;" Prof. Dr. Müller, Tübingen, "Pathologische Anatomie der tympanogenen Schwerhörigkeit;" Prof. Dr. Wullstein, Würzburg, "Die Tympanoplastik und ihre Resultate."

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

In order to complete the interrupted files of some foreign libraries, the ANNALS will buy from subscribers, at $1.50 per copy, a certain number of the following issues now out of print. Please communicate with the Business Manager, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, P. O. Box 1345, Central Station, St. Louis 1, Mo., U.S.A.

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OFFICERS

OF THE

NATIONAL AND INTERNATIONAL OTOLARYNGOLOGICAL SOCIETIES

VI International Congress of Otolaryngology

President: Arthur W. Proetz, M.D., St. Louis, U. S. A.
Secretary-General: Paul H. Holinger, M.D., 700 North Michigan Ave., Chicago 11, Ill., U. S. A.
Meeting: Washington, D. C., May 5-10, 1957.

International Bronchoesophagological Society

President: Dr. Theodor Hünermann, Düsseldorf
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: Philadelphia, May 12 and 13, 1957.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose Gros, Havana
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: Sixth Panamerican Congress, Brazil, 1958 or 1959

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, New York.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, Syracuse, N.Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

American Broncho-Esophagological Association

President: Dr. Clarence W. Engler, Cleveland, O.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: None, in 1957; San Francisco, Calif., May 21, 22, 23, 1958.

American Laryngological Association

President: Dr. LeRoy A. Schall, Boston.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Hotel Statler, Washington, D.C., May 3, 1957.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Dean M. Lierle, Iowa City.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: Section meetings only in 1957.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Gordon D. Hoople, M.D., Syracuse, N.Y.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.

American Otological Society

President: John R. Lindsay, M.D., Chicago.
Secretary-Treasurer: Lawrence R. Boies, M.D., University of Minnesota Hospitals, Minneapolis 14, Minnesota.
Meeting: Hotel Statler, Washington, D.C., May 4, 1957.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dean McAllister Lierle, M.D., University Hospital, Iowa City, Iowa.
Secretary: Michael H. Barone, M.D., 468 Delaware Avenue, Buffalo 2, New York.

Pacific Coast Oto-Ophthalmological Society

President: H. Leroy Goss, M.D., 620 Cobb Building, Seattle 1, Wash.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting: Coronado, California, April 7-11, 1957.

The Society of Military Otolaryngologists

President: Col. Wendell A. Weller, 3810 USAF Hospital, Maxwell AFB, Alabama
Secretary-Treasurer: Major Stanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.

Canadian Otolaryngological Society

President: Dr. G.M.T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. A. Henry, Medical Arts Bldg., Suite 328, Toronto, Ont.
Meeting: Banff Springs Hotel, June 17, 18 & 19, 1957.